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EXPERIMENTALLY INDUCED ANKYLOSIS OF THE STAPES.*†‡

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Research on human deafness has been hampered by the fact that no lower animal has ever been known to be subject to the disease of otosclerosis. Clinical manifestations of conductive deafness, the most common aspect of otosclerosis, are admittedly accompanied by demonstrable ankylosis of the stapediovestibular joint or the other ossicular joints.[1,2]

If experimental ankylosis could be induced, one further step in research on otosclerosis might be accomplished.

In the course of our investigations on the effects of various types of trauma to the region of the oval window, the authors decided to study the effect of an electric cautery needle applied to the area. The results of simple mechanical trauma were reported before the American Otological Society in May, 1958.[3]

Accordingly, the middle ears of eight cats were opened under nembutal anesthesia. The oval window and the stapes

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were clearly observed and mobility of the stapes verified in each case.

A Cameron Cauterodyne was used for the experiment. The Cameron Company states that this machine operates at a radiofrequency of 48 meters, and makes five million oscillations per second. "Cold" cutting and/or "cold" coagulation may be used. The depth of coagulation into the tissue is, according to the company, from $\frac{1}{4}$ of 1 mm. to 5 mm. respectively. "When using the cutting current at scalpel speed, healing by first intention may be expected, since only the lymphatics and capillaries are sealed with that type of cutting current. The coagulation is the type that causes no necrosis as may also be expected from the cutting current. No dehydration is formed beneath the coagulum with this type of current."

The needle used was completely insulated except at the tip, and coagulation occurred exactly at the tip. The effect was point-to-point, not diffuse.

The temporal bones of six cats (eleven ears) have now been processed and examined under the microscope. Postoperative time ranged from 24 hours to over six months. The results, in terms of ankylosis and of bone changes as a result of the touch of the cautery needle, which was set at "high," were so uniform and unusual that they merit a special presentation.

The reactions of the cats were observed following this procedure, and occasional movies were taken. No records of cochlear microphonics or electric potentials were taken. Crude observations were made in regard to hearing.

We produced ankylosis of the stapediovestibular joint in five of a possible eight ears. In two the stapes was removed from the oval window, and in one case death occurred the day after the second ear was operated upon. In some instances the incus became ankylosed to aditus and to malleus. In one instance incudostapedial ankylosis was effected. In three ears the stapedial crura were obviously replaced by fibrous tissue.

In addition to ankylosis, we have, in one ear, produced a greatly thickened and vascularized footplate of the stapes.

In all but two of the otic capsules we have produced microscopic changes which stimulate the histopathological picture of otosclerosis found only in the human ear.

Figure 1 illustrates the condition two-and-one-half months after the operative procedure. The cautery needle was applied to the anterior footplate and to the anterior crus. Con-

Fig. 1. Cat No. 16. Aut. 157, sec. 124, left. Postoperative time 2½ months. Ankylosis of the greatly thickened footplate is evident anteriorly. The fragile crura have been completely replaced by fibrous tissue. A charred mass (bone ash?) lies in the area. Arising from a hypervascular submucosa, vessels enter the footplate and a widened vascular channel in the otic capsule posterior to oval window. Anterior to oval window a widened vascular channel appears, surrounded by a counterstained area which simulates "blue mantling" in human otosclerosis.

siderable spread of coagulation over the upper posterior part of the middle ear was given to blanch and destroy the mucosa of the middle ear.

A postoperative note states that on the third day after the operation this cat was able to jump up on the table voluntarily and come down from the table normally.

The microscopic section reveals that no crura of the stapes are present. The footplate is firmly ankylosed anteriorly.

Within the otic capsule, both anterior to the oval window and posterior to it, there is an enlarged vascular channel with metachromatically stained bone surrounding the canal. No otitis media nor purulent labyrinthitis is present. In the dense fibrous tissue filling in the oval window niche in response to the trauma, a mass of charred particles is seen. This probably is the charred bone ash of the crura.

Fig. 2. Cat No. 13. Aut. 184, sec. 94, right. Postoperative time, 3 months, 5 days. Again we see ankylosis of the footplate and fibrous replacement of the crura. Anterior to the joint is a widened vascular channel lined with osteoblasts.

The organ of Corti exhibits a mound of cells of the usual contour and height throughout the cochlea. Outer and inner hair cells appear to have been disrupted in the lower coils and exhibit more nuclei than normal. Hensen's cells and the sustentacular, or Deiter's cells, show marked changes, the latter having lost their cytoplasmic membranes in the basal turn. Perhaps the excessive number of nuclei among hair cells is really due to displaced nuclei from the sustentacular cells. The apical organ is more nearly normal except for the sustentacular cell of the inner hair cell. Reissner's membrane and tectorium are *in situ* throughout. The latter is slightly

lifted from the limbus. Spiral ganglion cells appear normal but show a tendency to have the nucleus in the proximal end of the cell. There is hypertrophy of the tympanic nerve.

Figure 2 illustrates a similar condition with obvious ankylosis of the stapes and fibrotic replacement of the crura. Within the otic capsular wall an enlarged vascular area is

Fig. 3-a. Cat No. 14. Aut. 171, sec. 280, left. Postoperative time, 5½ months. Ankylosis is present both anteriorly and posteriorly. The footplate is thickened only near the ankylosed regions. Widened vascular channels with perivascular fibrosis occur both anterior and posterior to the oval window. A lymphadenitis was present in this ear.

seen, with the central region traversed by fibroblasts and the bony wall lined with osteoblasts.

The operative note in this case states that the cautery was applied in light amount to the footplate and to the anterior crus; also to the incudostapedial joint. The incudostapedial joint area has been completely replaced by fibrous tissue, as have the distal parts of the crura. Some evidence of the proximal parts of the crura remain at other levels than the one photographed. The organ of Corti presents the same microscopic picture as the preceding case with the exception

that Reissner's membrane is frayed and disrupted in one specific region in the middle coil, yet remains taut and *in situ*.

Figure 3-a presents a third case in which ankylosis has occurred both anteriorly and posteriorly. At these two areas the footplate is thickened. Widened vascular channels can be seen in the wall of the otic capsule, both anterior and posterior to the oval window.

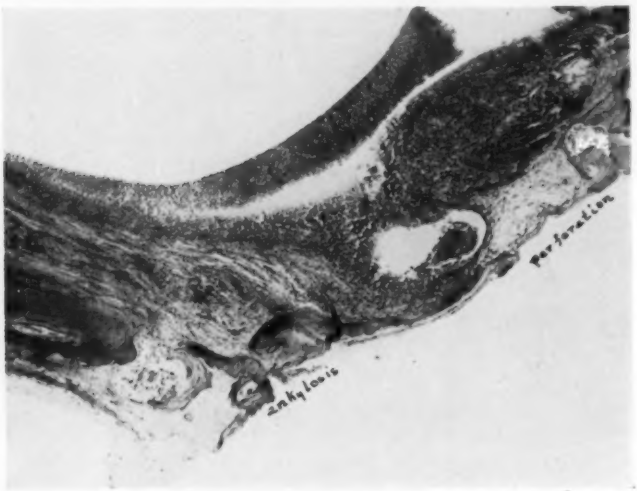

Fig. 3-b. Detail of the footplate of Cat No. 14. Aut. 171, sec. 260. This shows the area of perforation of the footplate. No bone covered this perforated area. Therefore, this footplate was not physiologically ankylosed.

The operative note on this ear states that the incus and crura were removed. A small perforation was made in the footplate. This perforated area did not close with anything but fibrous tissue, although this animal lived five-and-a-half months after the operation. Figure 3-b shows the persistent perforation. This stapes was, therefore, not physiologically ankylosed.

The cochlea in this case presents an organ of Corti of normal contour, but the structure definitely appears coagulated.

Spiral ligament of the lower basal turn is devoid of fibroblasts and connective tissue. Reissner's membrane sags over the limbus throughout; however, the spiral ganglion cells appear normal.

Figure 4 shows a detail of the peculiar and specific vascular reaction which this treatment seems to engender in the

Fig. 4. Cat No. 16. Aut. 157, sec. 95, right. Postoperative time, 3¼ months. Detail from the promontory showing metachromatic staining of bone around widening vascular channels. The left center field presents a picture similar to halisteresis. In other areas osteocytes are being set free by as yet unknown decalcifying processes. The freed cells are then converted either into fibroblasts, osteoblasts or osteoclasts.

otic capsule. A "blush" of purplish-red dye surrounds some of the vascular channels, while others remain normal in appearance. In these pathological areas decalcification has either already occurred or seems imminent. Clear areas along the border of the lesion suggest halisteresis. In these regions several osteocytes, recently freed from their lacunae, appear to congregate and form osteoclasts. These are really giant cells of the foreign body type. Their function must be to phagocytize fragments of the decalcified bone rather than to break it down, themselves. This seems to be the case, since

the clear halo with decalcification appears before the osteoclasts have actually formed.

Other freed osteocytes become converted into fibroblasts, filling up the space between vessel walls and bony walls. When the decalcification process has gone "far enough," *i.e.*, presumably to supply physiological local demands, osteocytes

Fig. 5. Cat No. 12. Aut. 175, sec. 199, right. Postoperative time 6 months, 9 days. Detail from another promontory showing widened vascular channels. The similarity to "blue mantling" in otosclerosis is striking in the longitudinal cut of the vascular channel. Fibrotic replacement of bone has occurred in the irregular cross section, above and to the right.

will then be converted to osteoblasts and start regenerating bone.

In the lower left field of Figure 4 we see the initial stage of the development of this type of pathological condition. A congested blood vessel is seen within the bony channel and on each side of the vessel are clear spaces. These spaces undoubtedly are formed by some fluid which has been exuded from the vessel. Inferiorly in this same small lesion, the effect of such extrusion upon the bone is evidenced by the elongate lighter spaces in the solid bony field.

Fig. 6-a. Ankylosis of footplate of stapes in a rat fed Lathyrus odorata by Dr. Lalich (University of Wisconsin). The head of the animal was processed in our laboratory at our request, to see if any evidence of stapedial ankylosis could be found in cases of nutritional disturbances.

Figure 5 illustrates a condition in another promontory. This was present six months and nine days after exposure to the cautery needle. The longitudinal section of the vessel shows the widened vascular channel with the surrounding

"blue mantling" which really acts like a chemical indicator, demarcating the pathological from the normal state of the surrounding bone. Under very high power the spread of this "indicator" can definitely be traced through the canaliculi.[4] In the center field, osteoblasts are forming along the bony wall of the vascular channel. To the upper right of center we see an irregular cross sectional area where fibrotic

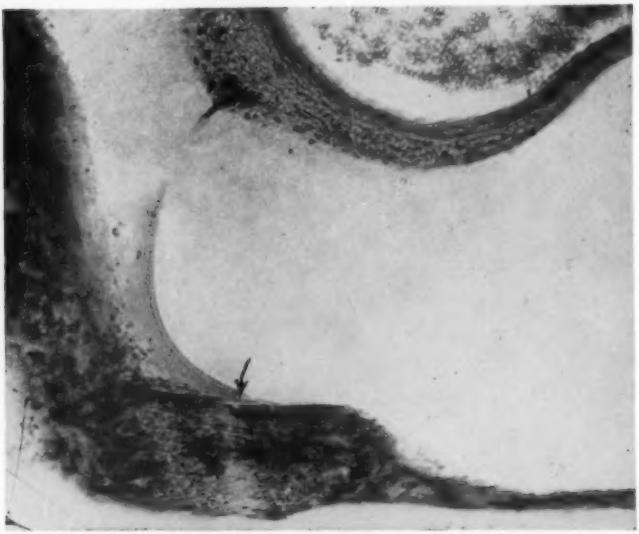

Fig. 6-b. Detail of calcium bar crossing the stapediovestibular joint space in the rat. This pathological calcium deposition is the result of nutritional disturbance. No middle ear infection is present. The cellular elements are blood corpuscles within the stapedial artery as shown in Fig. 6-a. This vessel is persistent in rodents.

replacement of bone is occurring. Here, also, osteoblasts and osteoclasts are developing.

These illustrations indicate that we have experimentally produced a lesion in the bony otic capsule of the cat, which simulates closely the histopathological lesion in the human otic capsule in cases clinically diagnosed as otosclerosis.

CONCLUSION.

Ankylosis of the stapediovestibular joint can be produced experimentally. This can be accomplished in one of several ways:

1. We have produced ankylosis of the stapediovestibular joint in five of eight possible cats' ears, by the use of electrocoagulation. The use of the electric cautery also produced lesions in the otic capsule, which simulate the histopathological picture of human otosclerosis.

2. We have produced ankylosis of the stapediovestibular joint by mechanical trauma, as reported at the American Otological Society in California in May, 1958. This was produced in four cats' ears.

3. The final illustration shows ankylosis of the stapediovestibular joint in a rat fed excessive doses of lathyrus factor by Dr. Lalich, of Wisconsin University. At our request we have processed several of the temporal bones of his experimental animals, and those of Iowa University. Evidence accumulates in our laboratory to the effect that altered nutrition may cause ankylosis of the stapediovestibular joint. Because of the prevalence of otitis media in these animals, we are awaiting publication of this last point until more examples without otitis media can be demonstrated. We are also feeding our own guinea pigs ground pea meal (Lathyrus sativa) with encouraging results.

The interpretation of our findings will require the investigative skill of a chemist and a biochemist. In producing the perivascular lesion in the bone we do not know whether we have an electrochemical effect or merely a thermal effect. We may have a true electrolysis of the bone. If so, what state of being in human pathology induces this kind of reaction in cases of otosclerosis? The potential lability of bone has been clearly demonstrated previously, but what is the chemistry of the tissue fluid which is able to wash out the bone salt so completely and yet so locally? These are imponderables which await further investigation.

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REGRESSION OF HEARING IMPROVEMENT AFTER MOBILIZATION OF STAPES.*†

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When stapes mobilization was introduced by Rosen in 1953, some otologists believed that it was not possible to mobilize an ankylosed stapes; others believed that once mobilized the stapes quickly would become fixed again. Since then considerable experience has been gained with this type of surgery, and it now generally is accepted that *most* fixed stapes can be mobilized with a significant improvement in hearing. With the passage of time, however, an impression has been gathering that the improvement in hearing obtained by stapes mobilization undergoes regression to the preoperative level in many cases. This study was undertaken to investigate the frequency and possible responsible factors of this postoperative regression in hearing.

Although the mobilization of the stapes operation has found a place in modern otological surgery, its true value can be established only after the long range benefits to the patient are appreciated. This study, however, is *not* based entirely on cases which have been observed for over very long periods of time. Due to the high percentage of regression which has occurred within the first few months after the operation, we believe it is important to indicate now the trend our cases is taking.

It was believed generally that growth in the otosclerotic lesion was responsible for refixation of the stapes and regression of hearing to the preoperative level. Our observations on

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experimental animals indicated that other factors may play a part in the refixation of the stapes.[1,2] In experimental animals mild surgical trauma at the stapedio-vestibular joint, injuring the adjacent stapes and vestibular wall initiated a very active tissue response. In this tissue reaction, hypervasculization and fibrosis, as well as the deposition of new bone, were observed. In many cases the stapedio-vestibular joint space actually was bridged by new bone and ankylosis of the stapes resulted.

It is reasonable to assume that a comparable tissue response could occur in the human, and the trauma induced in the process of mobilizing the stapes could partially be responsible for refixation of the stapes. In human surgery it has not been possible to examine the stapes of all cases in which a postoperative regression of hearing occurred; however, in all cases re-operated because there was regression of hearing, the stapes always was found reankylosed.

Our technique of mobilization has been modified from that used in the early days, when the mobilizing force was applied only to the neck of the stapes, to one in which the mobilizing force is applied directly to the lesion at the footplate. This orderly change in technique permits a convenient division of our cases into two groups. In the early group trauma to the footplate was minimal; in the other, some degree of trauma was induced in all cases as the forces for mobilization always were applied directly to the point of maximum fixation at the footplate. In this latter group the lesion was visualized with the microscope and classified into four groups according to the anatomic and pathologic findings.[3]

It has been noted that a higher percentage of cases had an immediate improvement in hearing when the forces for mobilization were applied directly to the point of fixation at the footplate. This apparently is a logical observation, for within any group of cases the otosclerotic lesion varies in extent from a minute area of fixation to a complete replacement of the footplate. There is a graduated scale in the size and extent of the otosclerotic lesion. Some stapes with a small area of fixation can be mobilized by any technique; in

other cases, the otosclerotic lesion is extensive, and our presently used techniques are useless. The percentage of good immediate results appears to increase with the amount of pressure that can be applied safely to the stapes; and most pressure can be applied directly at the point of fixation.

In the earlier techniques the strength of the crura was the limiting factor in mobilization of the fixed footplate. Only cases with small degree of fixation could be mobilized by applying pressure to the head or neck of the stapes. The stapedio-vestibular articulation on the other hand, was not traumatized directly in the process of mobilizing the stapes.

TABLE I.

Unclassified Cases—Force applied to head or neck of stapes.
Classified Cases—Force applied directly to footplate at lesion.

	Total Cases	No. Above 30 db	Per cent	Per cent Regression in 4 Mo.	12 Mo.	18 Mo.
Unclassified Cases	212	102	48	23.5	35.3	39.0
Classified Cases	142	85	60	36.0	.....	.....

With the application of force at the head and neck, 48 per cent of the cases obtained an immediate improvement in hearing above the 30 db level. Although no direct trauma to the stapedio-vestibular joint was induced in the process of mobilization, 23.5 per cent of cases were found to regress to the preoperative level within four months. The high rate of regression continued after this period, as 35.3 per cent had regressed within 12 months and 39 per cent within 18 months (See Table I).

Later cases which have been grouped in four classes according to the anatomic and pathologic findings can be studied in greater detail. In this group of cases the mobilizing forces were applied directly to the footplate at the point of maximum fixation, and an immediate improvement in hearing above the 30 db level was obtained in 60 per cent of cases. In this group of 142 consecutive cases, an unusually high number of cases happened to be of Class IV. Excluding this group of very unfavorable cases for mobilization by our present technique, 76.2 per cent of all others had an immediate improve-

ment in hearing to above the 30 db level. In our previous report[3] which included all Class IV cases, 72 per cent had an immediate improvement in hearing. Many more cases were mobilized when the force was applied directly to the lesion as the strength of the crura was not a limiting factor. More trauma, however, was induced at the footplate of the stapes in mobilizing cases with more extensive lesions. With the application of more force at the footplate, more trauma was induced to these structures. In the four months following the operation, 36 per cent of those cases which had an immediate improvement in hearing to above the 30 db level regressed to

TABLE II.

Regression of Hearing Improvement in Classified Cases Four Months Postoperative.

	No. of Cases	No. Above 30 db	% Cases Improved	Number Regress.	Per cent Regress.
Class I					
Early Otosclerosis	26	22	84.6	2	9
Class II					
Mod. Adv. Oto.	63	43	68	23	53
Class III					
Adv. Otosclerosis	21	16	76	4	25
Class IV					
Unfavorable Cases	32	4	12	2	50
Totals	142	85	60	31	36

the preoperative level. There were too few cases which were observed beyond the four-month period to make a significant report at this time.

The postoperative behavior of the four classes of otosclerotic involvement can be compared. In Class I were grouped cases in which the otosclerotic lesion was small. Palpation revealed fixation at one end of the stapes, while the other was free. The footplate was blue in color, and there was no narrowing of the stapedio-vestibular joint space. The otosclerotic lesion was not visible with the microscope. An immediate improvement in hearing was obtained in 84.6 per cent of cases. As the otosclerotic fixation was minimal, the mobilization procedure was not traumatic to the tissues at the footplate of the stapes. Only 9 per cent of this group showed regression to the preoperative level within four months (see Table II).

In Class II were grouped cases with visible narrowing of the stapedio-vestibular joint space by otosclerosis. The footplate was blue in color, but palpation revealed fixation at the involved end; the other end was free. The crura were not included in the otosclerotic mass. The majority of the 142 cases included in this study presented characteristics of Class II. In this class, the otosclerotic mass bridged the articulation and was so extensive that there was visible narrowing of the stapedio-vestibular joint space. Considerably more difficulty was encountered in mobilizing the fixed footplate. Head or neck application of force usually was not successful, as frequently the crura were fractured. Much more trauma occurred at the footplate of the stapes when the mobilizing forces were applied directly to the point of fixation. The adjacent vestibular wall, stapes and footplate were traumatized. When mobilization was achieved directly through the lesion by division of the otosclerotic mass the fracture permitted the roughened halves of the otosclerotic mass to oppose each other, leaving surfaces readily exposed to the tissue reaction which follows. Sixty-eight per cent of cases had an immediate improvement in hearing to above the 30 db level. Although this high percentage of cases had an immediate improvement in hearing, the rate of regression was also high, as 53 per cent returned to the preoperative level of hearing within four months (see Table II).

When the otosclerotic lesion was very extensive and involved, one end of the footplate including the adjacent crus, the case was characteristic of Class III. Here the footplate was of normal blue color except for the area near the otosclerotic involvement. The mobilizing technique had to be altered in this type of case as the lesion was too extensive for mobilization directly through the mass. The technique recommended by Fowler[4] is ideally applicable. The crurotomy avoids direct fracture through the otosclerotic mass, and the mobilization is made through normal bone with less trauma. Seventy-six per cent of the cases in this group had an immediate improvement in hearing to above the 30 db level. Twenty-five per cent returned to the preoperative level of hearing within four months (see Table II).

In Class IV were grouped cases unfavorable for mobiliza-

tion by our present techniques. In most instances, little could be done surgically, as the otosclerotic lesion had replaced the entire footplate or had reduced the oval window niche to a slit. In other cases the exposure was very unfavorable or the crura of the stapes were involved so that continuity of the ossicular chain could not be preserved. In this study of 142 consecutive cases an unusually large number of unfavorable cases occurred. This lowered the general average of good results considerably. An immediate improvement in hearing was obtained in only four cases of 32 in Class IV. In all four the footplate of the stapes, although completely infiltrated and thickened by otosclerosis, was able to be elevated and separated intact from the vestibule. Two of the four cases, however, regressed to the preoperative level within four months.

COMMENTS.

Regardless of the responsible factors, there is a surprisingly high percentage of cases which regressed to the preoperative level of hearing following the mobilization of the stapes. Although the active otosclerotic lesion may cause refixation of a mobilized footplate, evidence from animal experiments and analysis of our cases indicate that the tissue reaction following trauma induced by the mobilization procedure also may play a significant role.

This study has revealed that the percentage of regression in hearing following a successful mobilization of the stapes operation is proportional to the degree of trauma induced at the region of the stapedio-vestibular joint space in mobilizing the fixed footplate. The application of force to the footplate directly has elevated the percentage of immediate good results to above 70 per cent.

The technique of mobilization used in these cases has been conservative, and an attempt has been made to leave the stapedio-vestibular joint space as normal as possible following the mobilization of the stapes. Regression in hearing has occurred regardless. There are many cases which have maintained hearing above the 30 db level for over three years without any sign of change. This, of course, attests to the value of this surgery. As the high percentage of regression

is a serious problem, our attention should be directed toward improving the technical approach in order to reduce the post-operative regression in the improved hearing.

CONCLUSIONS.

Of all cases having an immediate improvement in hearing above the 30 db level, 36 per cent were found to regress to the preoperative level within four months. This high rate of regression is probably due in part to the refixation of the stapes by an actively growing otosclerotic lesion and in part by the tissue reaction, which occurs as a result of the trauma induced in mobilizing the otosclerotic lesion at the footplate.

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THE EIGHTH BRAZILIAN CONGRESS OF OTOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

The Eighth Brazilian Congress of Otolaryngology and Bronchoesophagology will be held in Porto Alegre, Brazil, the first week of September, 1959. President: Dr. Ivo A. Kuhl, Andradas 1727, Porto Alegre, Brazil.

TUMORS OF THE GLOMUS JUGULARIS.

A Report of Nine Cases.*

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Guild,[1] in 1941, reported the presence of small structures in human temporal bone sections located near or in the wall of the jugular bulb, to which he gave the name "glomus jugulare," and in his paper in 1953,[2] he further discussed these bodies. He had noted that they occurred with almost equal frequency along the course of the nerve of Jacobson. The number of glomus bodies in an ear varied from possibly none to as many as twelve. Of 88 ears studied, 60 had either one, two, three or four glomera each. About one-half were found in the adventitia of the dome of the jugular bulb; one-fifth in the osseous canal, through which the nerve of Jacobson enters the middle ear from the jugular fossa, and about one-fourth had a glomus formation in the mucosa of the cochlear promontory as far peripherally as the descending facial canal.

Simpson and Dallachy[3] reviewed the reported cases of tumors of the glomus jugularis, and found a total of 172 cases, only 14 of which had been reported prior to 1945. Ages in the 172 cases varied from 22 years to 69 years, and it was noted that the tumor ocurred five times more frequently in women than in men. These authors state, "Tumors of the glomus jugulare in the middle ear grow slowly, and the constituent cells are uniform in appearance. There is no capsule, however, and continuous expansion of the growth occurs with extension outward along the external canal, upwards into the middle cranial fossa, medial with damage to or destruction of the structures of the internal ear and labyrinth, or downwards into the jugular foramen. Spread may occur

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in more than one direction. The lesion in the vast majority of recorded cases shows locally invasive characteristics."

They further stated that, "while the site of origin largely determines the direction in which the growth spreads, the cases largely fall into four main clinical groups. Under their Group I classification there are aural symptoms only. In Group II there is neurological involvement developing years after the aural symptoms. The nerves involved by pressure are usually the IXth, Xth, XIth, and XIIth. In Group III the neurological symptoms and aural symptoms develop concurrently. The growth here probably arises near the origin of the tympanic nerve. In Group IV, the least common, the neurological symptoms precede the aural symptoms.

Except for Group IV, of which there were no cases, each of our nine cases fits aptly into one of the foregoing classifications. In Group I, the largest, there were six cases. Of these six, four were Negro, two were white, and sex was equally divided between male and female. Ages ranged from 31 years to 65 years.

A case, a white female, aged 23, came under the Group II category. In Group III, both patients were white, female, and ages 37 and 43 years respectively.

GROUP I.

Case 1—P.G. (024592), female, white, aged 55.

Initial visit Sept. 7, 1949. Complaint of thumping sensation in left ear, noted since preceding November, which was synchronous with the heart beat. There was an occasional pain in the left ear. Examination revealed a redness of the inferior quadrant of the left tympanic membrane. Hearing test showed 15-20 db loss of hearing, left; mastoid X-rays were negative. No diagnosis established.

Return visit May 2, 1951. No treatment since previous examination 20 months previously. Tinnitus was persistent in left ear, and an increased sensation of fullness of the left ear had been noted for several months. Examination revealed a reddish mass at the inferior quadrant of the left middle ear; hearing test showed a loss of 30 db in the left; X-rays of mastoid, petrous apex, and base of skull were negative. A tentative diagnosis of glomus jugularis tumor was made; surgery, however, was postponed.

Return visit May 24, 1952. No apparent enlargement of tumor; no particular increase in patient's symptoms; biopsy was done. Pathological report: Glomus jugulare tumor; treatment: X-ray, 3000 *r* units.

Return visit June 16, 1953. Tumor was still present; no progressive

enlargement nor increase in symptoms was noted; treatment: X-ray, 3000 *r* units.

Yearly observations were made during 1953 to 1958.

Last progress visit Sept. 17, 1958. Patient had no particular complaints other than a very mild tinnitus in the left ear. Examination revealed slight thickening of the left drum; a small red tumor was visible through the drum, apparently arising from the floor of the middle ear. This tumor appeared to be smaller than before; hearing remained about the same as before, possibly slightly improved (see Fig. 1).

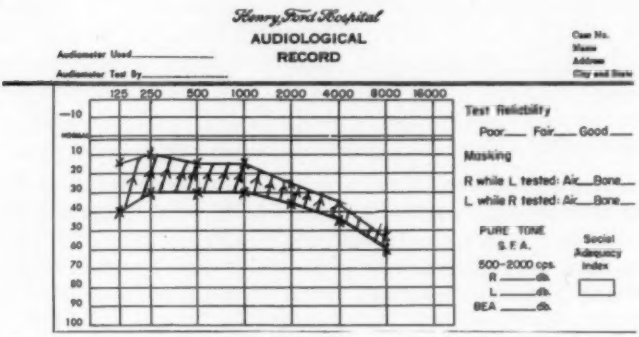

Fig. 1. Case 1, No. 024592—Hearing tests left ear, 1951 and 1958. Slight improvement of hearing noted.

COMMENT: After a period of ten years, this patient is alive and well, with very few symptoms relative to the tumor in the left ear. This would not appear to be active and the patient should, in all probability, outlive the growth in her middle ear.

Case 2—E.K. (828721), male, white, aged 31.

Initial visit April 3, 1956. Complaint of thumping sensation, right ear, which had been present about eight months. This was continuous and was synchronous with his heart beat. Examination revealed a normal right ear drum, with a reddish mass noted in the middle ear arising from the floor of the middle ear; hearing test revealed approximately normal hearing.

Surgery April 27, 1956. The tumor was removed through the auditory canal with elevation of the drum, as in the stapes mobilization procedure. Considerable bleeding was encountered, which was readily controlled. Pathological diagnosis: Glomus jugulare tumor (see Fig. 2).

Last progress visit Sept. 11, 1958. Patient was entirely well with no ear symptoms two-and-a-half years following removal of tumor. There was no evidence of recurrence of the tumor on examination; slight improvement of hearing was noted.

Case 3—J.A. (768230), male, Negro, aged 32.

Initial visit Oct. 13, 1954. Complaint of thumping sensation, left ear, more marked on lying down; two months in duration; no hearing loss was noted. Examination of the left auditory canal revealed a mass or

heaped-up area in the auditory canal just external to the drum. This mass involved the floor of the canal and extended upwards on the posterior wall; it was soft to the touch and appeared to be cystic; a mass of red tissue in the middle ear behind the inferior quadrant of the drum was also noted; both seemed to be continuous. X-rays of mastoid and middle ear appeared normal; audiogram showed apparently normal hearing. Biopsy was taken from floor of canal; there was slight bleeding. Pathological diagnosis: Glomus jugulare tumor.

As patient was from out-of-town, X-ray therapy was given by his local physician; this apparently had no effect on the tumor, and the patient became increasingly aware of his heart beat, and felt that the thumping sensation was likewise increasing.

Feb. 22, 1955, four weeks since X-ray therapy. No change in size of tumor; thumping sensation, left ear, increased.

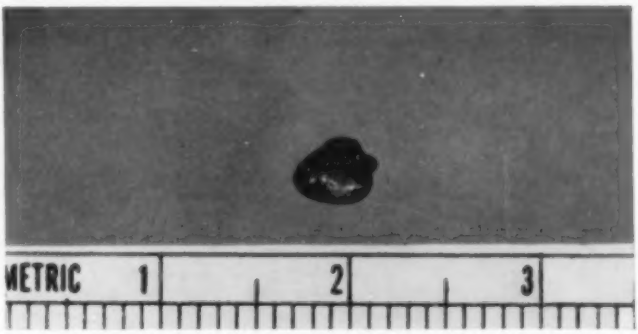

Fig. 2. Case 2, No. 828721—Photograph of tumor (encapsulated) removed from middle ear.

Progress visit July 2, 1955. There appeared to be some enlargement of the tumor.

Surgery July 12, 1955. Radical mastoidectomy was done; the tumor was removed, and no excessive bleeding was encountered; recovery was uneventful.

No evidence of recurrence of tumor in left ear or mastoid on Dec. 30, 1957. Cavity well healed; audiogram showed a loss of hearing (see Fig. 3).

Last contact, November, 1958. Patient has remained well, with no tinnitus and no symptoms other than a hearing loss. This contact by letter was four years after initial visit.

Case 4—E.K. (673952), female, Negro, aged 65.

Initial visit March 22, 1952. Patient was first treated in the Emergency Room because of severe epistaxis; the episodes of epistaxis had been recurrent over a three-year period, and were attributed to her known hypertension. Her past history indicated that in 1926 she had noted a "buzzing and bubbling" sensation in the left ear followed by pain. Paracentesis of the left drum resulted in considerable bleeding, and the

ear had continued to discharge. Since 1945 a progressive weakness of the left side of the face had been noted. Because of her ear symptoms and findings, she was referred to the Otological Division and seen on the same day. Examination revealed swelling of the left mastoid, pinna and pre-auricular area and purulent discharge in the left auditory canal. Tumor growth was seen filling the auditory canal; there was complete paralysis of the left side of the face. Chest X-rays were negative; mastoid films revealed that "Left mastoid shows sclerotic changes visible in all portions of the mastoid area; no actual bone destruction. The diameter of the left auditory canal is much smaller than on the right; does not look like bone tumor."

Surgery, April 1, 1952. Mastoidectomy was done on the left; the mastoid cortex was intact, but bled very freely; the mastoid cells were destroyed and filled with a reddish, infected tumor mass; there was

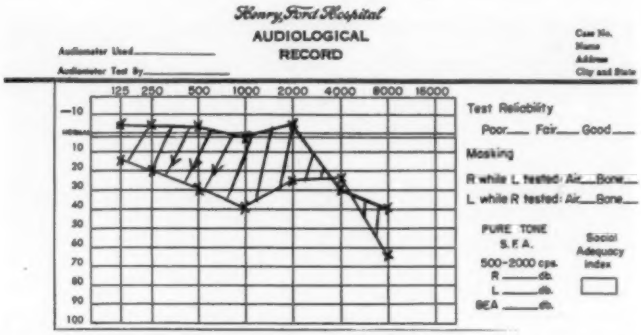

Fig. 3. Case 3, No. 768230—Initial and final hearing test, left ear, reveals a loss of hearing.

complete destruction of the mastoid and bony ear canal with extension of the tumor into the parotid gland anteriorly. The middle ear was filled with tumor mass; because of bleeding, the tumor could be only partially removed. Pathological report: Glomus jugulare tumor. On examination of the nasopharynx, we discovered that this tumor was prortuding from the orifice of the Eustachian tube.

On April 8, 1952, patient was discharged from the hospital with a well-healed incision. X-ray therapy was advised; but the patient refused further treatment, and we have had no further contact since that time.

COMMENT: This is a patient with a slow growing glomus jugulare tumor, which remained localized to ear and surrounding tissues; symptoms were reported to extend over a 26-year period, with a facial paralysis of seven years' duration. We presume patient is dead.

Case 5—M.B. (834046), female, Negro, aged 60.

Initial visit June 12, 1956. Complaint of impaired hearing in right ear; hearing loss had been noted since 1937; there had been no pain or tinnitus. Examination revealed a redness of the lower half of the right drum, suggestive of a mass in the middle ear. Audiogram showed an air conduction loss for all tones in the right ear and a bone conduction loss

for the high tones in the right ear. Chest X-ray was negative; mastoid X-ray showed normal development and aeration of the mastoid cells with no destruction.

Surgery, June 12, 1956. Under general anesthesia, the tumor was removed through the auditory canal. The drum was elevated as in the stapes mobilization operation; profuse bleeding was encountered. After removal of the tumor, the ear drum was replaced in its normal position and the auditory canal was packed. Pathological report: Paragangioma or glomus jugulare tumor.

Progress visit Sept. 4, 1956. Perforation of right drum noted; no tumor noted; complains of marked tinnitus in right ear.

Last contact in November, 1958. Patient reported by letter that tinnitus was still present; hearing has been unchanged. No report of recurrence of tumor.

Case 6—J.D. (903415), male, Negro physician, aged 40.

Initial visit Feb. 3, 1958. Complaint of impairment of hearing, 20 years, left ear, with pulsating sensation and high-pitched tinnitus in that ear. Following a paracentesis of the left drum in 1954, the ear had drained for two years but has since remained dry. Patient was concerned about possibility of carcinoma, left ear. Examination revealed a large, bluish mass which appeared to be protruding from the inferior three-fourths of the left drum. Audiogram revealed a combined deafness of approximately 70 db.

Examination under general anesthesia Feb. 5, 1958. On examination, the cyst ruptured; it had appeared to be a large bluish, cystic structure, which seemed attached to the floor of the canal in the region near the annulus, and which contained a brownish fluid. After opening the cyst wall, there was noted lying within and beneath, a bluish-red, friable and extremely vascular soft tissue mass; biopsy was taken. Pathological report: Glomus jugulare tumor.

Treatment Feb. 10th to March 13th, 1958, X-ray therapy, 4000 *r*.

Progress visit April 2, 1958. Vestibular function was markedly diminished, left ear.

Surgery April 30, 1958. Radical mastoidectomy, left, was done, revealing a large pneumatic mastoid. There was tumor filling the entire middle ear and extending into the Eustachian orifice, the epitympanum, and the mastoid antrum. This tumor was completely removed, except for vestiges over the jugular bulb and carotid artery.

Progress visit June 26, 1958. Mastoid cavity and ear looked clean; audiogram showed 80-90 db hearing loss (see Fig. 6).

Progress visit Sept. 4, 1958. Ear dry; skin graft healed. No recurrence of tumor.

COMMENT: Seven months after surgery, the mastoid is clean, and there is no evidence of recurrence.

GROUP II.

Case 7—I.B. (524432, female, white, aged 23.

Initial visit Nov. 10, 1947. Complaint of pain around right ear for previous three weeks; cough, and loss of 14 pounds of weight in the previous six weeks, and of having "generally felt bad all over" for two years. History revealed that she had had a plugged-up sensation in the right ear, which had begun during a pregnancy five years earlier. Two years previously she had had an acute infection of the right ear and

since, has had an occasional discharge; general physical examination was essentially negative. The auditory canal on the right was clean, but the drum was red and appeared swollen in its posterior quadrant, with a heaped up area along the canal floor close to the drum. Mastoid X-rays were negative. Biopsy was done and was reported as showing squamous cell papilloma. On treatment as an infection, her pain disappeared and there was much less discomfort.

Next progress visit 15 months later. Complaints were concerning her throat, primarily an inability to swallow for three days. Examination revealed a jugular foramen syndrome, with involvement of the IXth, Xth, and XIIth cranial nerves. The right ear drum was red, and there was a reddish heaped-up area on the floor of the canal against the drum, which appeared papillomatous. X-rays revealed slight hazing of the right

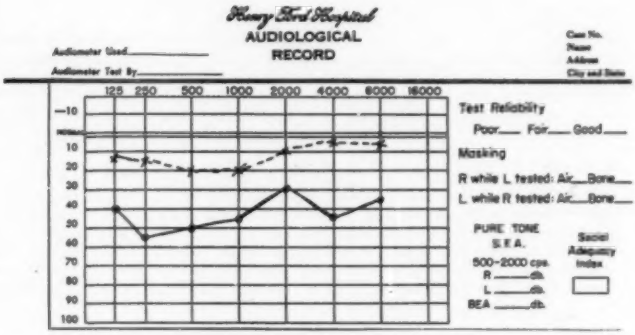

Fig. 4. Case 7, No. 524432—Mild hearing loss, 20 db, left ear, conductive loss. Right ear: hearing loss of approximately 40 db, conductive loss.

mastoid; the base of skull was negative; the petrous ridges were smooth in appearance, and there was no evidence of destruction.

Progress visit Feb. 21, 1949. Audiogram revealed approximately 40 db loss of hearing, right ear (see Fig. 4).

Surgery Feb. 23, 1949. A radical mastoidectomy was done on the right. The cortex was removed, and serous discharge pulsated from the mastoid cells. The cell walls appeared soft but were intact; there was a reddish tumor mass in the middle ear projecting from the floor of the middle ear and extending into the region of the jugular bulb. (This was removed as far as possible.)

Pathological report: Glomus jugulare tumor.

Surgery, May 21, 1949. Right mastoid was reopened. The floor of the auditory canal and middle ear was removed; the facial nerve was isolated and the tumor exposed; this could not be removed because, despite pinching off the carotid, there was such profuse bleeding. The patient developed a facial paralysis and a left hemiplegia.

Treatment Aug. 5, 1949: X-ray therapy, 2000 *r* right ear and mastoid.

Patient was seen at regular intervals during 1949 to 1954. The facial

paralysis improved, as did the left hemiplegia, although there was not complete recovery from the latter.

On March 31, 1954, X-rays of the mastoid region revealed a progressive destruction of the mastoid and adjacent bone. Treatment: Cobalt therapy, 4000 *r*.

Last contact May 6, 1958. Patient alive and fairly well; some evidence of right facial weakness and left hemiplagia remained. Mastoid was well healed; no evidence of tumor in mastoid or middle ear; still suspect presence of intracranial progression, but no definite evidence to support this.

GROUP III.

Case 8—T.K. (727175), female, white, aged 37.

Initial visit Aug. 26, 1953. Complaint was of hoarseness; however, history elicited the following: In June, 1952, following a tonsillectomy, she noted increasing hoarseness with associated poor appetite. In November, 1952, difficulty in swallowing developed, though there was no regurgitation. There also was noted a weakness and pain in the left shoulder. At the same time, there was noted a throbbing and pounding in the left ear. Examination revealed an area of marked redness in the inferior quadrant of the left drum. Audiogram showed a conductive deafness of approximately 15 db in the left ear. Caloric test was normal in each ear. Neurological examination disclosed a paralysis of IXth, Xth, XIth and XIIth cranial nerves. A tentative diagnosis of glomus jugulare tumor was made.

Treatment Nov. 21, 1953: X-ray therapy, 2400 *r*, over a three-week period.

Patient has been followed at intervals from 1953 to 1958. She has shown a definite decrease in her neurological symptoms without the development of any new symptoms.

Progress visit Feb. 12, 1958. There was still slight deviation of tongue to the left. Voice had improved.

On Sept. 30, 1958, patient felt her voice was good. Solid foods were swallowed without difficulty, although there was an occasional slight difficulty with fluids. On examination, a small pinkish blush was noted behind the left drum inferiorly, so small as to be easily missed. The tongue still protruded slightly to the left and atrophy of the left side was still present. Movement of the soft palate seemed normal; there was fixation of the left vocal cord and arytenoid. Audiogram revealed a slight improvement in hearing.

COMMENT: No biopsy was taken, so the tentative diagnosis of glomus jugulare tumor was never substantiated. X-ray therapy seems to have arrested the growth. This patient has been under observation for more than five years.

Case 9—E.K. (680217), female, white, aged 43.

Initial visit May 28, 1952. Seen by Division of Neurosurgery. Complaint of severe throbbing headache, worse on the left, continuous, and aggravated by turning head to the left. Had associated aching pain in the left shoulder and neck, vague in onset, but present six to eleven weeks; deafness and a pounding noise in the left ear had been noted at about the same time. Weight loss of 10 pounds associated with anorexia; had occurred in previous four weeks. Neurological examination revealed mild papilledema; no nystagmus. There was minimal left facial weakness, atrophy of left side of tongue, protrusion of the tongue

to the left, hyperactivity of reflexes on left and limitation of lateral movement of neck to the left; some decrease of hearing on left side was noted.

Our otological consultation elicited a history of tinnitus and deafness, left, for approximately five months. Examination revealed an intact left tympanic membrane, which was discolored, and with a peculiar fullness noted in the upper and posterior quadrants; audiogram revealed a bilateral combined deafness of 20 db in the right ear and 55 db in the left (see Fig. 5). X-ray showed an extensive lytic lesion in the base of the left petrous. Impression: Tumor, left middle ear; involvement of VIIth, VIIIth, IXth, and XII cranial nerves.

Surgery, June 6, 1952. Sub-occipital decompression revealed a reddish gelatinous tumor eroding through bone; the tumor infiltrated the petrous and occipital bones; this tumor bled freely. A biopsy was taken; ligation of the external carotid and occipital arteries was done, but this did

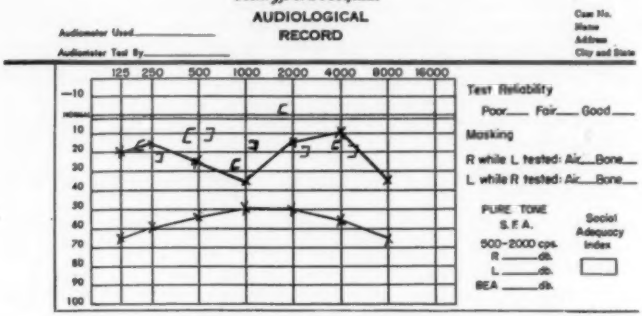

Fig. 5. Case 9, No. 680217—Combined deafness, bilateral. More marked loss, left ear.

not seem in any way to affect the vascularity of the tumor. Pathological report: Glomus jugulare tumor. Treatment: X-ray therapy was given over a three-week period.

Patient's course was gradually down-hill. There was an increasing enlargement of the tumor in the left middle ear, an increase in the facial paralysis, and increased difficulty in swallowing, which developed from an apparent extension of the tumor around the brain stem. A bilateral VIth nerve paralysis also developed in the late stages.

Patient expired Jan. 9, 1953. No autopsy was obtained.

COMMENT: This patient was admitted on Neurosurgical service with a cerebellar tumor of apparently short duration. There was involvement of the VIIth, VIIIth, IXth, and XIIth cranial nerves; an intracranial operation revealed a glomus jugulare tumor which apparently had extended from the left middle ear.

TREATMENT AND RESULTS.

Of our nine cases, three patients had surgery only:

1. 828721, Case 2, male, white, aged 31. Symptoms localized to ear, eight months in duration. Tumor of middle ear removed through drum by endaural approach. Two-and-one-half years later, patient is well with no evidence of recurrence.

2. 673952, Case 4, female, Negro, aged 65. Ear symptoms for 26 years. Tumor involved mastoid, auditory canal, middle ear, and parotid gland, with extension into Eustachian tube. Because of the extent of its growth and the bleeding, the tumor could not be entirely removed. No further treatment or follow-up. Patient presumed dead.

3. 834046, Case 5, female, Negro, aged 60. Symptoms limited to ear, 19 years in duration. Tumor removed through drum by endaural approach. No evidence of tumor recurrence two years later. Has perforation of ear drum, persistent tinnitus and nerve deafness.

Three patients had a combination of surgery and X-ray.

1. 768230, Case 3, male, Negro, aged 32. Symptoms, two months in duration, limited to the ear. No apparent decrease in tumor size as result of X-ray therapy. About seven months later, radical mastoidectomy with removal of tumor. No symptoms four years later except a hearing loss. No evidence of recurrence of the tumor.

2. 903215, Case 6, male, Negro, aged 40. Symptoms, 20 years in duration, limited to the ear. X-ray therapy given, followed about seven weeks later by radical mastoidectomy and removal of growth. Nine months post-operatively, there is no evidence of recurrence.

3. 524432, Case 7, female, white, aged 23. Symptoms, five years in duration, limited to ear at initial visit. Fifteen months later, neurological symptoms were present, involving the IXth, Xth, and XIIth cranial nerves. Radical mastoidectomy with incomplete removal of tumor. Mastoid reopened three months later. Tumor could not be removed because of excessive bleeding which could not be controlled.

Three months later, X-ray therapy, 2000 *r*; five years subsequently, X-ray therapy, 2700 *r*; one year subsequently, Cobalt therapy, 4000 *r*.

Patient alive 11 years later, with no evidence of tumor in ear or mastoid. Suspicion of intracranial involvement remains

Two patients were treated with only X-ray therapy.

1. 024592, Case 1, female, white, aged 55. Seen in 1949 and no diagnosis established; diagnosis made three years later; local symptoms in ear of four years' duration.

In 1952, X-ray therapy, 3000 *r*; in 1953, X-ray therapy, 3000 *r*.

Patient alive and well 10 years after onset; very small tumor still present in the middle ear. Has a mild tinnitus but no further loss of hearing. It is my opinion that this lady, now 65 years of age, with no evidence of progression of the tumor, will outlive her tumor.

2. 727175, Case 8, female, white, aged 37. Neurological symptoms at onset. Ten months later when first seen, there was paralysis of IXth, Xth, XIth, and XIIth cranial nerves. Tumor, left ear, with jugular foramen syndrome. Normal vestibular test. Diagnosis of glomus jugulare plausible. No biopsy obtained. Diagnosis not proven. Patient is definitely improved five years later. A small mass, very much smaller than it was initially, was noted in the left middle ear. Voice is better; paralysis still present, slight difficulty in swallowing fluids.

One patient on Neurosurgical service had intracranial surgery and X-ray therapy.

1. 680217, Case 9, female, white, aged 43. Admitted on Neurosurgery

with intracranial symptoms. History of severe throbbing headache, worse on left. Aching pain in left neck and shoulder of six to eleven weeks' duration. Deafness and pounding in left ear noted about same time. Cerebellar extension of tumor. On suboccipital decompression, glomus jugularis tumor found. X-ray therapy given, but course was gradually down-hill with enlargement of tumor in the left middle ear, increase in facial paralysis, bilateral VIth nerve paralysis and extension of tumor around brain stem. Patient expired approximately six months after initial contact.

SUMMARY AND CONCLUSIONS.

1. A report is made of nine cases of tumor arising from the glomus jugulare. Eight cases have been proven, one case has a presumptive diagnosis because of the location of the tumor in the middle ear and the sequence of symptoms.

2. The majority (eight) of these tumors were slow growing; however, one patient with cerebellar symptoms had an apparent rapid onset, and a rapid downhill course (Case 9).

3. In spite of the duration of symptoms in several cases, not one of our cases has been reported as malignant.

4. The symptoms produced by these tumors depend on the site of origin and their extension. In six patients, the tumor appeared to have its origin in the middle ear; two had neurological and aural symptoms develop at approximately the same time, and one patient developed aural symptoms, followed a few years later with neurological symptoms.

5. The duration of symptoms varied considerably. The majority had symptoms of rather short duration; however, three patients (all colored) had symptoms of 19 years, 20 years, and 26 years. It is of interest to note that these three tumors which had their origin in the middle ear, had only local extension of the tumor into the adjacent areas, and they revealed no evidence of malignancy in spite of their long duration.

6. The age of onset (judged from the history) varied from 18 years of age to 51 years of age. In seven cases, the symptoms first became apparent in the third and fourth decade; three patients first noted their symptoms at 18 years, 20 years, and 51 years of age, respectively.

7. Six patients were female, three were males, a ratio of

two females to one male. Five of our patients were white, four were colored.

8. Five patients with symptoms and tumors limited to the middle ear, who received either surgery, X-ray, or a combination of both, are alive with no evidence of recurrence of their tumor.

9. X-ray therapy appeared to be beneficial in our patients but has not eradicated the tumor. It seems to shrink the tumor and to lessen the bleeding. Two patients who received only X-ray therapy appear to be improved with no evident progression of their symptoms. Three patients whose surgery followed X-ray therapy have no evidence of recurrence of their growth.

10. Bleeding has not been a serious complication except in two cases. At surgery, removal of a rather extensive growth was attempted and produced excessive bleeding with inability to remove the growth entirely in these two patients.

11. Of nine patients with a tumor of the glomus jugulare, six are alive and well with no evidence of recurrence of their tumor. One patient is alive (has had four pregnancies since onset of her tumor) with no local evidence of recurrence, but there is a suspicion of intracranial extension. One patient died of cerebellar extension. One patient is presumed dead.

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FACIAL PARALYSIS; A METHOD OF TREATMENT WITH MASSIVE DOSES OF HISTAMINE.

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The purpose of this paper is to present a preliminary report of the results of treatment of facial paralysis with Histamine Diphosphate by intra-muscular injection of massive doses. Twenty cases were treated; 13 cases of Bell's palsy, or idiopathic paralysis, and seven cases of herpes zoster oticus.

The generally accepted etiology of the majority of Bell's palsies is an ischemia of the facial nerve near the stylomastoid foramen, caused by arteriospasm. These views are held by Kettel[4] and others. As a result of this ischemia, various structural changes take place in the facial nerve between the lateral semicircular canal and the stylomastoid foramen.

The extent of these alterations depends upon the severity of the interruption of the blood supply. The nerve becomes unduly constricted at the stylomastoid foramen and edematous proximal to this point, while tiny hemorrhagic streaks are seen running longitudinally within the sheath. The swelling of the nerve within the fallopian canal results in compression of the adjacent blood and lymph vessels, and consequently a vicious circle arises. Collier[1] states that a reversible ischemic block may be present for long periods in the nerves of the limbs without leading to permanent change, while the same block occurring in the rigid facial canal will produce increasing vascular stasis and edema leading to complete degeneration. She further states that a mixed lesion may occur, some of the axons undergoing degeneration, while others remain in a condition of reversible block and are capable of recovering. This is the key to the understanding of the effect of decompression. Sullivan[2] also agrees that disturbance of the blood supply is the first cause of this palsy,

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but thinks that the paralysis is a result of the pressure upon the nerve following edema in the rigid canal.

Medical treatment includes heat, massage and electrotherapy. Drugs with vaso-dilator effects have been used, such as nicotinic acid, intravenous histamine, and papaverine. Symphatholytic drugs have also been tried, such as ergotamine tartrate and tetra-ethylammonium chloride. Lately, Cortisone has been proposed, and good results were reported by Robbins.[6] Cervical sympathetic block has been proposed.

Surgical treatment consists of decompression of the nerve. Tickle's[5] indications for decompression surgery included: *1.* cases of acute Bell's palsy that have shown no improvement within two months; *2.* cases where recovery is partial and no further improvement shown in two to three months; *3.* cases of paresis in which several attacks have occurred causing considerable psychic trauma to the patient. Kettel[4] says many authors rely completely upon response to faradic stimulation, claiming that a complete recovery will never occur when the response is negative. He says that the majority of his cases showed no recovery after two months of observation and hence indicated surgical intervention. Since 75 to 85 per cent of all ischemic facial palsies recover under medical treatment he does not consider any operation at the onset of palsy. Kettel also feels that decompression will prevent relapse and recurrence of palsies.

As to prognosis, nearly all authors agree that from 75 per cent to 90 per cent of these cases will recover spontaneously. Response to faradic stimulation (Tickle[5]) is important and where this response is maintained for two weeks, nearly all cases will recover. If no improvement occurs within two months, surgery is indicated. Lierle[3] says that in typical Bell's palsy prognosis is good for at least partial recovery in a period of a week to several months, but complete recovery is rare. In cases of paralysis of five months' duration or longer, where there has been no improvement of the paralysis one can expect very little improvement after a decompression operation.

The second group of cases are those of herpes zoster oticus.

FIG. 1—BELL'S PALSY.

Patient	Onset	Interval to Treatment	Duration Treatment	Symptoms	Treatment Given	Final Result	Return of Function
1. F.J., M. (35)	12-27-50	2 days	11 days.	Paralysis (left), complete; pain and burning.	Histamine (IM) 1-10,000; Vitamin B Complex (IV).	Complete function.	11 days
2. A.C., F. (15)	1-29-52	2 days	6 months.	Paralysis (right), complete; severe earache at onset.	Histamine (IM) 1-10,000	Complete function.	6 months
3. L.P., M. (60)	3-30-52	1 day	3 months.	Paralysis (left), complete; severe earache.	Histamine (IM) 1-10,000	Complete function.	3 months
4. J.G., F. (40)	9-11-53	2 weeks	1 month.	Paralysis (right), severe neuralgia (right) face and ear.	Histamine (IM) 1-10,000; Riboflavin 10 mg. orally (q.i.d.)	Complete function.	1 month
5. T.C., M. (49)	9-21-54	2 weeks	1 month.	Paralysis (right), complete.	Histamine (IM) 1-10,000, Vitamin B Complex orally.	Complete function.	1 month
6. J.P. (15½)	10-51	3 years	17 months continuously and at 2-month intervals to date.	Complete paralysis with pulled face (left); severe headaches.	Histamine (IM) 1-10,000; Vit. B Comp. Tab. 1 b.i.d.; patient had headache reactions.	Expression normal; Function returned 10%.	17 months
7. C.H., M. (54)	2-17-55	2 weeks	4 months.	Paraesthesia anesthesia, pain (right) with complete paralysis.	Histamine 1-10,000; sedation.	Complete function.	1 month

8. F.H., F. (32)	3½ years prior to treatment, started treatment 6-27-55		2 months.	Paralysis complete with some pulling left face.	Histamine 1-10,000; patient had severe reaction headache.	Complete function.	2 months
9. A.K., F. (50)	2-5-54	2 weeks	2 weeks.	Paralysis left complete; pain, burning.	Histamine 1-10,000, 3 times weekly for 6 doses.	Complete function.	2 weeks
10. D.W., F. (51)	1-2-57	3 weeks	2 months.	Paralysis, left, complete with severe pain region Vth and VIIth nerve.	Histamine 1-10,000, 1.0 cc. daily; had already received Vit. B, steroids, foradic stim. and nerve block—no results.	Complete function.	4 weeks
11. M.O., F. (46)	12-10-56	4 weeks	4 months and continues to present.	Paralysis complete, right; severe pain; severe edema eyelids.	Patient in severe auto accident with development paralysis in 1 wk.; nerve block (cervical); Vit. B Comp. and steroids; no result.	90% function.	4 weeks
12. G.B., F. (32)	9-25-57	4 weeks	2 months.	Paralysis complete, left.	Histamine 0.5 cc. increased to 1.0 cc. q. 3 days.	Complete function.	4 weeks
13. T.S., F. (52)	4-12-57	11 days	5 weeks.	Paralysis complete, right.	Histamine 0.5 cc. to 1.0 cc. q. 3 days.	Complete function.	7 weeks

According to Gill[7] the disease is an acute involvement of the skin characterized by thick-walled groups of vesicles which may affect the pinna and extend into the external auditory canal, or vice versa. Pain and discomfort are associated with the disease and may precede the vesicles. These vesicles may recede, and scar after an acute phase of seven to ten days. When facial and auricular nerve palsies occur simultaneously, the term Hunt's syndrome is used; the affection is rare. Treatment consists of orthodox lotions, such as ammoniated mercury, boric acid and salicylic acid cleansing of the skin, and pituitrin injections in some cases. Day and Jordan advise that recovery in cases of herpes zoster oticus is rare.

The 20 cases herein presented were treated by intramuscular injection of Histamine Diphosphate (Abbot No. 3286, .275 mgm. per cc.) of 1 to 10,000 dilution, using 1.0 cc. at daily intervals until return of function began. Then injections were given every two or three days until recovery was complete. Maintenance dose was carried on at one month intervals for the next six months. At that time therapy was discontinued.

Since each case presented rather individual characteristics in symptoms and progress, charts are prepared to cover the entire series. Figure 1 covers all the cases of Bell's palsy. The usual pertinent data as to age, sex, predominant symptoms and interval from onset of symptoms to the time therapy began, is charted. Cases Nos. 1 and 3 were of only several days' duration when seen, and hence the question of spontaneous recovery must always be considered.

Case No. 2 is a high school girl in whom symptoms persisted for six months. Decompression was delayed and intensive treatment continued as outlined above, with complete return of function in six months. Of interest is the fact that this patient became senior queen of her class, following recovery from her paralysis.

It can be assumed that in the remaining cases in which the paralysis was present from one to three months prior to

treatment, recovery would probably not occur spontaneously in all cases.

One case, No. 8, is worthy of special mention, because of the duration of paralysis prior to treatment. A 32-year-old female was first seen on June 27, 1955, with left paralysis complete for three and one-half years. Treatment was carried on as outlined above, and complete selective nerve function returned after two months' treatment, and has remained functional since then.

Another case, No. 6, a 15½-year-old male, had paralysis, complete for three years prior to treatment. Recovery has been only partial, but the facial contour is good in the "at rest" position without any "pulling" or "drooping" of the face.

In summarizing the results of this therapy in 13 cases:

1. Duration from onset of symptoms to treatment was from two days to three years.

2. Duration of therapy from onset to beginning return of function varied from two weeks to six months.

3. One case has not completely recovered after 17 months' treatment.

In reviewing the literature, this author was able to find only two cases of Bell's palsy treated in a similar manner. Skinner,[11] in his report of 19 cases, treated these two cases with Histamine Diphosphate in a 1/10,000 dilution. His remaining 17 cases were treated with 1/10,000,000 dilution of Histamine with good results. He expressed a preference for the low dosage therapy.

The only other reference to treatment with histamine was by Loomis,[10] who used it by intravenous injection in five cases, all with good results.

In Figure II are included seven cases of herpes zoster oticus. All presented symptoms of severe pain, vesicles and other sensory losses such as sense of smell. Interestingly, six of the seven were females. Under treatment by the same methods, recovery was uniformly good in all cases, even

FIG. 2—HERPES ZOSTER OTICUS.

Patient	Onset	Interval to Treatment	Duration Treatment	Symptoms	Treatment Given	Final Result	Treatment to Cure
14. H.J., F. (33)	9-5-49	3 days (9-8-49)	7 months.	Vesicles (AD); severe pain; weeping ear.	Hist. 1-1 million Penicillin (IM) Lextron Cap. Niacin 50 mgm. (severe reaction)	90% function.	3½ months
15. F.H., F. (41)	2-28-51	3 weeks (3-22-51)	1 year reg.; infrequent checkups with booster since.	Vesicles (AS); severe pain.	Hist. 1-1 million 1-100,000:1-10,000 Ref.: Dr. Day, Dr. Jordan.	90% function with "lag".	6 months
16. M.G., F. (40)	11-27-52	2 weeks (12-11-52)	4 months with return checkup and booster.	Vesicle (AS) lip; shingles left body.	Hist. 1-10,000; later tonsillectomy and dental ext.	Compite function.	3 months
17. E.S., F. (64)	12-27-54	1 week	5 months.	Severe shingles left forehead, left ear, left face and weeping; severe pain.	Hist. 1-10,000; Aureomycin Oint. sedation; cath. Eust. tube later.	Complete recovery paralysis and mod. scar face, ear lobe.	2 months
18. C.S., F. (62)	12-23-55	10 days (1-3-56)	6 weeks.	Vesicles (AS) complete; severe pain; loss of smell; mult. perforations; entire drum denuded.	Hist. 1-10,000; sedation; (severe reaction).	Complete function.	2 weeks
19. M.K.B., F. (23)	1-24-56	3 weeks	4 weeks.	Severe pain ear; rt. vesicles, ear lobe and face burning; paralysis complete, right.	Hist. 1-10,000 daily; Aureomycin Oint. to face.	Complete function.	4 weeks
20. G.R., M. (45)	6-20-56	4 weeks	3 months.	Paralysis complete left vesicles, ear lobe, drum and canal, left; sudden pain.	Hist. 1-10,000 daily.	Complete function.	4 weeks

though scarring persisted in the external auditory canal and face in case No. 17. Since most authors agree that despite meager literature on the matter, prognosis is very grave in such cases under present methods of therapy; perhaps histamine offers its greatest usefulness in herpes zoster oticus.

In conclusion, 20 cases of facial paralysis have been reviewed under therapy with histamine by intramuscular injections in massive dosage. Return of function has been uniformly good in a short period of time. In none of these cases did any disturbing side effects or complications arise from the drug administered.

Histamine in massive doses offers a rapid and simple method of treatment for Bell's palsy and herpes zoster oticus.

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IVALON AS A NASAL SEPTAL IMPLANT.*

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Experimental and clinical evidence indicates that living autogenous cartilage grafts are preferable to preserved isogenous cartilage when used to correct nasal deformities.[1] A third and least desirable choice is to utilize a non-absorbable foreign substance in rebuilding the nose; however, a search for a suitable substance is justifiable if the end-results outweigh the hazards involved when such material is implanted in living tissue, in this instance, the nose.[2] When such substances are utilized, the clinician should make a careful study of what happens to the material in the nose; how living tissue reacts to such an implant in that anatomical location, and the functional and cosmetic results attained. He is further obligated to report his findings in order to further enhance our knowledge in this important phase of nasal reconstructive surgery. This is a report of such a study made on seven cases in which Ivalon (Polyvinyl) sponge was utilized as a nasal septal implant.

Two problems encountered in nasal septal surgery prompted this study. One deals with the question of the need for replacing the cartilaginous and bony components of the septum removed during the course of a submucous resection. Many operations have been devised to preserve as much of these structures as possible.[3] We endeavor to employ such procedures when feasible, but situations do arise, when most of the cartilaginous and portions of the bony septum have to be sacrificed. A suitable implant must be inserted to strengthen the septum, prevent cicatricial contraction, maintain the stability of the external nose, and to eliminate the "sail flapping" effect that occasionally occurs when only mucous membrane comprises the septum.[4,5] The second problem concerns the

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prevention of septal perforations following surgery. It is a complication which is a constant annoyance to the patient because of crusting, excessive drying, bleeding and occasional voice changes which may occur. Closure of such perforations is difficult.[6] Perforations occur in spite of the most meticulous technique. Atrophic mucous membrane, marked deformity of the septum, drying and crusting due to abnormal course of air currents, and trauma to the septum from repeated cauterizations can create situations in which perforations are inevitable. Should a perforation occur, the operator must provide a bed upon which the mucous membrane can grow; otherwise the healing membranes on each side will meet and a permanent perforation result.

The answer to the two problems may lie in the procurement of suitable material to use as septal implants. Autogenous and isogenous septal cartilage grafts are the most desirable. We have a cartilage bank for preserving cartilage removed at operation, but the segments are too small to serve as a septal implant for the purposes outlined. Autopsy material is difficult to procure, and the specimens removed are not much better than the material obtained at operation. We were attracted by the work being done by Dr. John J. Kralik, head of the Department of Cardiovascular Surgery at Marymount Hospital, on the use of Ivalon sponge in experimental cardiac surgery on dogs, and in its use in the human heart. After studying his results, we decided to use the material as a nasal septal implant. We found in the literature scant references to the use of Ivalon in nasal surgery.[2,7,8,9]

Ivalon surgical sponge is a white polyvinyl alcohol sponge made by treating foamed polyvinyl alcohol with formaldehyde.[10] The result is a fine-textured white sponge which is soft and resilient when wet, and hard when dry. If remoistened, it regains its original softness. It can be easily cut with scissors or a sharp knife. It can be sterilized by boiling in water and autoclaving. On heating in water some permanent shrinkage does occur, but once having shrunk further shrinkage does not occur on resterilization. It has been shown that when introduced into the body, the body fluids enter the sponge, and fibroblasts and blood vessels

grow into it. In reality the Ivalon sponge acts as a framework for living tissue. It is claimed that a foreign body reaction does not take place, and no untoward complications had occurred in laboratory animals.

We prepared Ivalon for use in the nose in the same manner as for the use in dog experiments and in human hearts, since no untoward reactions have been observed when the material had been treated in this fashion. A suitable segment was cut from the block, thoroughly rinsed in sterile distilled water, then compressed in a metal form consisting of two plates held together by four thumb screws. The metal compressor containing the Ivalon was then sterilized in an autoclave for one half hour at 250°. The Ivalon segment was then removed from the compressor and packed in a cloth and resterilized, then stored for future use. If stored for over 30 days, the pack was again resterilized before being used. If Ivalon was needed in the course of an operation, it was first moistened in sterile saline, then cut and trimmed to the desired shape. After moistening, the average thickness of the segment was 2 mm. Care was exercised in inserting the material to be sure no cartilaginous deflections or bony ridges remained, which would cause buckling or displacement of the implant. A prominent anterior nasal spine was usually the most troublesome. Whenever possible we tried to avoid a transfixion incision in order to have one side of the septum intact, for experience had shown that the line of incision was the avenue of escape for the implant if it was extruded. We likewise attempted to maintain the integrity of the membranous septum, for we believe that its proper function is important in facial expression. Vaseline packing was routinely inserted in both sides of the nose. In dealing with septal perforations we found that drying by inspired and expired air was a factor in healing. The longer the packing could be kept in the nose, (up to seven or eight days in some cases), the more likely would healing of the perforation occur. We differed in one respect from the procedure followed in cardiac surgery, in that antibiotics were not routinely administered pre and postoperatively. The latter cases were considered as infected when the Ivalon was inserted into the heart, and large doses of Penicillin were given in each case. Since, as will be dis-

cussed later, we did not feel that infection was the chief cause of complications, we saw no justification for using antibiotics as a routine procedure.

The number of cases was small, but these were carefully studied and observed almost daily during the initial period following the operation. The longest period of observation has been two years and three months, and the shortest period has been two weeks. The number was small because Ivalon was implanted only in those cases where extensive portions of the bony and cartilaginous septum had to be removed and could not be replaced or transposed because of marked deformity, and in those cases where through and through tears had occurred during the course of the operation, and the complication of a permanent perforation was feared. We did not wish to use a foreign substance in the human nose, about whose reaction we knew little, without justification for doing so. Prior animal experimentation, using Ivalon implants in the nose had not been feasible. Direct observation of the effects of Ivalon implantation upon the dog heart and the human heart, and references in the literature led us to believe that Ivalon was not injurious to human tissue. The cases in which Ivalon was used were not selected prior to operation. The judgment as to whether or not Ivalon would be implanted depended upon what transpired at the time of operation, *i.e.*, if large segments of the bony or cartilaginous septum had to be removed, or if tears in the mucous membrane occurred. The ready availability, size and ease of handling Ivalon made it a superior implant to an isograft or any other foreign body substance with which we were acquainted.

REPORT OF CASES.

Case 1. R. T., a 53-year-old white female was referred to us because of a deformed nose and difficult nasal breathing due to an injury sustained in an auto accident three years ago. Examination revealed an external nasal deformity associated with a marked deflection of the nasal septum.

Laboratory examination showed a white blood count of 6,700, hemoglobin 10.5 gms. and hematocrit of 33 per cent. Blood sugar was 109 mgm. per cent and blood urea nitrogen 8.5. Urine was negative for albumin and sugar.

A submucous resection combined with a rhinoplasty was done on

October 5, 1956. Most of the cartilaginous septum and a portion of the bony septum had to be removed. An Ivalon implant was inserted to provide a support for the dorsum of the nose.

She was given Penicillin 800,000 units daily for two days, then Terramycin 250 mgm. three times a day for the following eight days. The vaseline packing, which had been inserted into both sides of the nose was removed on the fifth postoperative day. The cosmetic and functional results were good. The Ivalon implant was apparently well tolerated, and when the patient was last seen in January of 1959, no change was noted in the nose.

Case 2. A. W., a 32-year-old white male came to us with a complaint of nasal blockage associated with crusting and dryness of the nose. Examination revealed a marked displacement of the nasal septum to the left with spur formation and obstruction of the nasal airway. The patient showed clinical and laboratory evidence of nasal allergy.

Laboratory studies showed a white blood count of 7,600, a hemaglobin of 15.5 gms., and a hematocrit of 50 per cent. Urine was negative for albumin and sugar.

A submucous resection of the nasal septum was done on August 28, 1955. At operation the mucous membrane of both sides of the cartilaginous septum was very thin and atrophic, and a through and through tear occurred in spite of the meticulous technique. An isogenous cartilage graft from the cartilage bank was inserted between the mucous membrane flaps in the area of the tears in order to provide a bed for the mucous membrane of both sides of the septum for the purpose of preventing a permanent perforation.

No antibiotic was given postoperatively, and the vaseline packing which was inserted in both sides of the nose was removed the second postoperative day. In spite of daily postoperative care, the isograft sloughed out completely on the tenth postoperative day and a perforation resulted. Because of annoying bleeding and crusting, it was decided to attempt a closure of the perforation using an Ivalon implant.

The operation was performed on September 15, 1956, one year and ten months after the first operation. The perforation measured 2 cm. in length and 1 cm. in height. The mucous membrane flaps were carefully separated, and an Ivalon implant measuring 3 cm. by 2 cm. was inserted and held in place with vaseline packing.

Again, no antibiotics were given. The packing was removed on the second postoperative day. The mucous membrane edges would not heal, and the Ivalon implant sloughed out on the fourteenth post-operative day. A permanent perforation has resulted. The tissue reaction to the Ivalon was moderate and was characterized by edema of a moderate degree and crusting.

Case 3. E. L., a 57-year-old white male was referred to us because of recurrent attacks of epitaxis and obstruction to breathing. He had had cautery done to both sides of the septum elsewhere. Examination revealed a marked deflection of the nasal septum to the left. The bleeding point was difficult to localize because of the septal deformity. A submucous resection of the septum was advised.

Laboratory findings on admission showed a white blood count of 7,450, a hemoglobin of 15 gms., and a hematocrit of 49 per cent. Urine was negative for sugar and albumin.

The operation was performed on August 28, 1956. During the course of the operation a through-and-through tear occurred in the mucous membrane at the site of the previously done cauterizations. An Ivalon

implant was inserted between the mucous membrane flaps to provide a bed upon which the edges of the torn membranes could heal.

Penicillin in the amount of 400,000 units, twice daily, was given for two days and the packing removed on the second postoperative day. The mucous membrane completely bridged the perforation in the right side of the nose but did not heal over on the left side. The Ivalon presented itself at the line of incision which had been made in the left vestibule and had to be removed finally through the unhealed line of incision, ten weeks after the operation. The implant was easily removed, indicating that there was little if any fixation due to ingrowth of adjacent living tissue. The line of incision promptly healed, and the septum was intact.

Case 4. D. B., a 15-year-old white female was referred to us because of an external nasal deformity and difficulty in breathing following an injury to her nose in childhood. Examination revealed a deformed and depressed nasal bridge associated with a deflected septum, which completely obstructed the airway on the left. A submucous resection was advised, to be followed two years later with a rhinoplasty.

Laboratory findings on admission showed a white blood count of 7,800, a hemoglobin of 12 gms., and a hematocrit of 37 per cent. Urine was negative for albumin and sugar.

The operation was performed on August 31, 1956. The cartilaginous septum was so badly deformed and fragmented that most of it had to be removed. An Ivalon implant was inserted between the mucous membrane flaps and held in place with 2-0 chromic catgut mattress sutures.

Penicillin, 400,000 units, once daily was given for three days. The vaseline packing was removed on the second postoperative day. The Ivalon was apparently well tolerated and the single incision made in the left vestibule healed; however, ten weeks after the operation the Ivalon implant presented itself through the old line of incision and had to be removed four months after implantation.

The implant was readily removed, indicating little if any fixation by infiltration of living tissue. The line of incision promptly healed.

Case 5. A. K., a 49-year-old white male was referred to us because of a deformed nose and obstruction to breathing, due to a recent fracture of the nose superimposed on an old healed fracture. Examination revealed an external nasal deformity associated with an obstructing deviation of the septum to the right. A submucous resection and rhinoplasty was advised.

Laboratory findings on admission showed a white blood count of 8,100, a hemoglobin of 12 gms., and a hematocrit of 38 per cent. Urine was negative for sugar and albumin.

The operation was performed on October 4, 1957. The destruction and deformity of cartilage and bone of the septum made it necessary to insert an Ivalon implant to aid in stabilizing the nose. The usual transfixion incision had been made. The implant was fixed with 3-0 chromic catgut mattress sutures.

No antibiotic was given. The packing was removed on the fourth postoperative day. The transfixion incision healed on the right side of the septum but persisted on the left. An inflammatory reaction with a mucopurulent drainage, pain and edema set in and the Ivalon implant protruded through the incision one month after the operation. The drainage persisted until the Ivalon was removed, ten weeks later. The implant was readily removed indicating little, if any, fixation by infil-

tration of living tissue. The inflammatory reaction quickly subsided and the line of incision healed.

Case 6. T. A., a 23-year-old white male was referred to us because of difficulty in breathing through the nose, following a football injury in his high school days. Examination revealed a marked deflection of the nasal septum to the left with obstruction of the airway. A submucous resection of the septum was advised.

Laboratory findings on admission showed a white blood count of 9,600, a hemoglobin of 13 gms., and a hematocrit of 44 per cent.

The operation was performed on November 17, 1958. A transfixion was made. Since most of the cartilaginous septum had to be removed an Ivalon implant measuring 2 cm. by 5 cm. was inserted and anchored with 3-0 chromic catgut mattress sutures.

Penicillin, 600,000 units and Streptomycin 0.5 gms. was given daily for four days. The nasal packing was removed on the third postoperative day. The postoperative course was uneventful.

Two weeks following surgery, it was noted that the left nasal vestibule became occluded by marked hyperplasia of the soft tissue of the columella, making it difficult for the patient to breathe. Since the swelling did not subside the patient was taken to surgery for a revision operation on January 3, 1959, two months after the initial surgery. It was decided to remove the Ivalon with a portion of the adjacent mucous membrane though it was apparently being well tolerated. This was done for the purpose of histologic study.

It was noted that the mucous membrane peeled off easily from the underlying Ivalon implant. In fact, a segment of mucous membrane had to be sutured to the underlying Ivalon before a representative specimen could be obtained. The histologic findings will be presented later. Recovery was uneventful.

Case 7. J. P., a 43-year-old white male was referred to us because of nasal blockage. Examination revealed a deflection of the nasal septum blocking the right nasal airway. A submucous resection of the nasal septum was advised.

Laboratory findings on admission showed a white blood count of 6,100, a hemoglobin of 12.9 gms., and a hematocrit of 42 per cent. Urine was negative for albumin and sugar.

The operation was performed on December 29, 1958. Because of the amount of septal cartilage removed an Ivalon implant measuring 2.5 cm. by 5 cm. was inserted and sutured in place with 3-0 chromic catgut sutures.

Patient was placed on Aureomycin 250 mgm. every six hours and 600,000 units of Penicillin daily for three days. The packing was removed on the second postoperative day and the patient discharged. The following day, he presented himself with nasal obstruction due to marked edema of the nasal mucous membrane of the septum. He showed no systemic evidence of infection and had no complaints other than the obstruction. Aspiration needling failed to yield pus or old blood. He was finally taken to surgery one week later and the Ivalon implant removed. Cultures taken from the bed where the implant laid showed few coliform bacilli and a few Staphylococcic aureus of the hemolytic type. The implant was removed with ease. The edema gradually subsided. Patch tests on the forearm made with fresh Ivalon and segments of the removed Ivalon specimen showed no skin reaction after 24 and 48 hours.

Two additional cases of through-and-through tears in the nasal mucous membrane occurring at the time of operation warrant inclusion in this series though Ivalon was not used. In both instances isografts from our cartilage bank were implanted in the septum to provide a bed for the growth of the epithelial tissue. One occurred in a young man of 17 and the other in a 47-year-old adult. In the former patient the center of the graft necrosed and a small permanent perforation resulted. In the latter patient the entire graft sloughed out, and a large perforation resulted. In Case 2 of the Ivalon series an isogenous nasal cartilage graft used at the time of the original operation sloughed out making a total of three cases in which isografts failed to prevent the formation of perforations.

In summary seven cases ranging from 17 to 57 years in age were presented. The Ivalon implant has been retained in one case for over two years. One implant which was apparently being tolerated well was removed for histologic study. The implant was rejected by the host tissue in five cases. Only in one case could true infection be demonstrated. In five cases the Ivalon was implanted because of extensive removal of cartilage and bone from the septum. In two cases it was implanted to prevent permanent perforations. Antibiotics were not routinely used nor given in large doses as in heart surgery. Five operations were submucous resections alone and two were rhinoplasties combined with submucous resection. Three additional cases (including Case 2 in the original series of seven), in which isografts from a cartilage bank were used to prevent perforations were appended. In all three cases the isografts were rejected totally or partially and permanent perforations resulted.

DISCUSSION.

At this point in the presentation the casual reader might hastily conclude that Ivalon is unsuitable as a nasal septal implant since in five and possibly six of seven cases the Ivalon was not retained to date (in Case 6 an apparently well tolerated Ivalon implant was deliberately removed for histologic study). There is a wise old saying to the effect that we

learn from our failures, not our successes. We had to ask ourselves the question why to date Ivalon was rejected in five (or possibly six) out of seven cases when implanted in the nose, yet was well tolerated, as far as could be determined in 35 cardiac implants in the human heart (Dr. Kralik's series). We also had to ask ourselves the question why human nasal cartilage isografts utilized in three cases appended for com-

Fig. 1. Microphotograph of a dog's heart removed ten days after Ivalon implantation.

parative study were likewise rejected in whole (two cases) or in part (one case).

The apparent failures for the implant to be accepted cannot be attributed to faulty technique as we followed the usual procedures we did in routine submucous resections and rhinoplasties, nor upon infection, for in only one case was a mild infection demonstrated. The answer as to why the Ivalon implant was accepted in the heart and not in the nose must be attributed to something in the inherent nature of the two respective tissues. To study this problem further we enlisted the aid of Dr. J. Kralik, who furnished the heart specimens

obtained from dogs which had been sacrificed at varying periods after Ivalon had been implanted in the animals. He also made available to us a specimen of a human heart from a patient who died of congestive heart failure 30 days after Ivalon was implanted to cover an atrial septal defect. Dr. E. Siegler, Director of Laboratories, very kindly consented to do the histologic studies.

Figure 1 is a photomicrograph from a slide of the heart of a dog sacrificed ten days after Ivalon was implanted to close

Fig. 2. Microphotograph of a human heart removed at autopsy 30 days after Ivalon implantation.

an artificially produced inter-atrial septal defect. The endocardial surface is to the left. One notes an exuberant, acute inflammatory reaction extending from the endocardial side into and between bundles of Ivalon. This reaction under higher magnification consists of fibrin, red cells and polymorphonuclear leukocytes and edema fluid.

Figure 2 is a photomicrograph of a section from a human autopsy heart examined 30 days after Ivalon was implanted

to close an atrial septal defect. In the area photographed, which is at the junction of the Ivalon and the auricle, one notes the usual constituents of granulation tissue, namely, fibrin, proliferating fibroblasts, round cell infiltration and newly formed capillaries and veins. At about the center of the photograph, the straight line relationship between the Ivalon on the left and the auricle on the right is no longer seen. There is a foreign body type of giant cell reaction

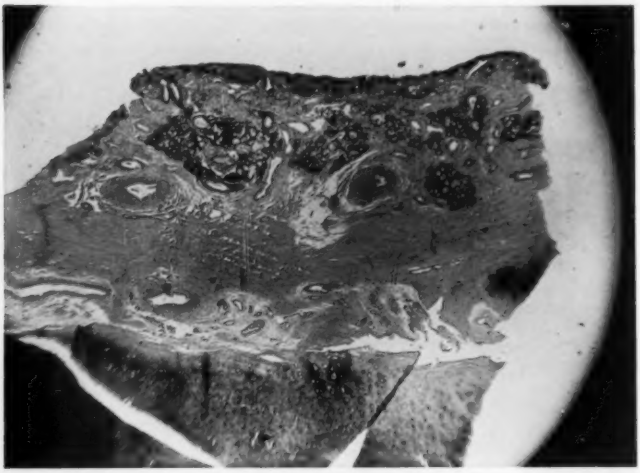

Fig. 3. Microphotograph of a normal human septum taken under low magnification.

present immediately adjacent to some of the Ivalon fibers. The most important finding demonstrated is the extensive infiltration of the granulation tissue into the interstices of the Ivalon. In the central portions of the Ivalon only fibrin and inflammatory cells are seen. Presumably the fibroblasts have not had time to move in.

Figure 3 is a low power view of a normal human nasal septum. At the top of the photograph is non-keratinized squamous epithelium. In the lamina propria one found the usual mucous and serous glands. Arteries and veins are

present in this layer as well as in the perichondium layer. A layer of cartilage is seen at the bottom of the photograph.

Figure 4 is the most significant microphotograph in this series. It shows a section of nasal septal mucous membrane adjacent to Ivalon implanted into a patient's nasal septum and removed 60 days following surgery. The photo illustrates why the entire implant was removed with relative ease. Unlike the hearts, the Ivalon to the left has not been infiltrated

Fig. 4. Microphotograph of a section of Ivalon covered by nasal mucous membrane. Specimen removed 60 days after implantation of the Ivalon.

to any extent by granulation tissue. In other words, the straight line relationship between the sponge and the nasal tissue has been retained almost completely. An occasional foreign body giant cell is present at the junction of the lamina propria and Ivalon. Only a small amount of granulation tissue is found in the interstices.

Figure 5 shows one of the few large foci of granulation tissue invasion seen in the center of the Ivalon specimen.

Figure 6 is a photomicrograph from a slide of the heart of

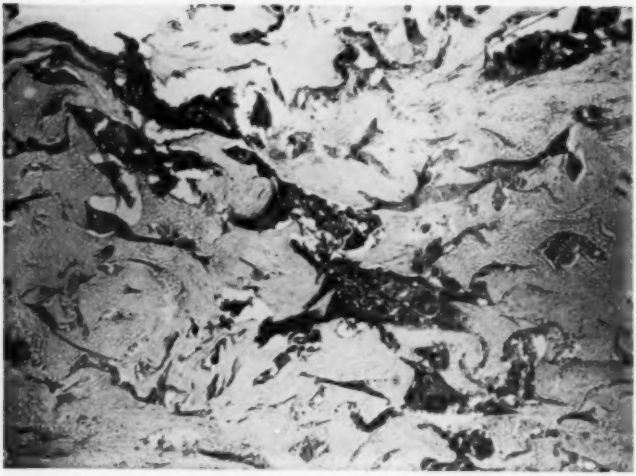

Fig. 5. Microphotograph of a focus of granulation tissue in the center of the Ivalon specimen.

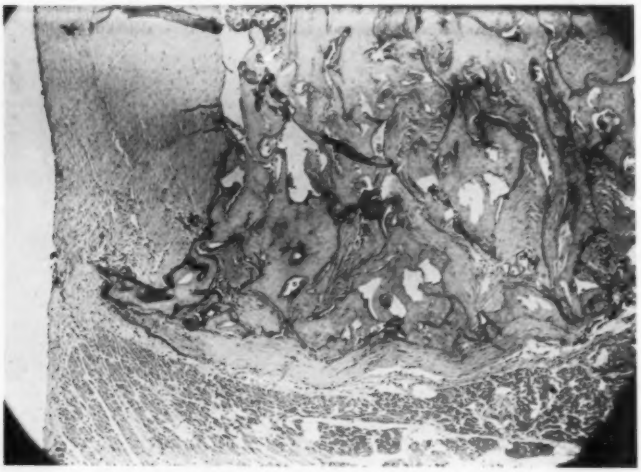

Fig. 6. Microphotograph of a dog's heart removed 90 days after Ivalon implantation.

a dog sacrificed 90 days after Ivalon was implanted to close an artificially produced inter-atrial septal defect. The Ivalon sponge occupies the field at the top of the photograph and normal myocardial fibers are seen at the bottom of the picture. Dense, hyalinized fibrous tissue has formed a complete capsule around the sponge and can be seen to have infiltrated the sponge to an extensive degree. The vertical band to the left is one of the endocardial layers. This is the type of end result one would like to see in all artificial implants.

We do not feel that the differences in degree and type of reaction between what occurs in the human and dog heart as compared to what happens in the human nose, which were so graphically illustrated in the microphotographs, can be explained merely on the basis of variations in the blood supply, the presence of a barrier such as the perichondrium, or upon environmental factors such as drying currents of air. We feel that another and perhaps more potent factor might be that of local tissue immunity, or, as we would prefer to express the term, local tissue resistance.

Williams, in speaking of lethal granulomatous ulceration, stresses the marked localizing powers of skin and mucous membrane.[11] The midline facial tissues may possess greater localizing powers than the tissues in other parts of the body, in this instance the heart. Kahn, in discussing tissue immunity and its possible relationship to midline facial granulomatous ulceration makes the pertinent observation: "Apparently the exposed tissues of the body carry a tremendous burden of defense. Through evolutionary ages these tissues have had the task of warding off micro-organisms and preventing their entrance into the blood stream and into deeper tissues. It is this great burden that these tissues carry that is unquestionably related to allergic disturbances. Like over-vigilant guards, the surface cells begin to treat substances as though they were harmful micro-organisms and react with great intensity on slight provocation."[12] Wilson and Miles, in speaking of body defense mechanisms, conceive a type of immunity confined to one particular area of the body or to one particular kind of tissue.[13] Conley in discussing the principle of cooling as applied to the composite graft in

the nose attributed the gratifying results he attained by utilizing this method to diminution of the biological activity demands of the graft until normal physiological processes had attained the level of survival.[14] In our opinion an equally valid explanation might be that the defense mechanism of the recipient site may have been depressed by the cooling to such a degree that the graft had an opportunity to "take."

SUMMARY.

A report was submitted on the fate of Ivalon which was implanted into the nasal septum of seven individuals. Three additional cases in which human cartilage isografts were utilized for the same purpose were appended. In five and possibly six of the cases in which Ivalon was used, the implant was rejected by the nose. The implants were rejected in whole or part in all three cases in which isografts were inserted.

Histologic studies of tissue reaction to Ivalon in the human and dog hearts were made and compared to the reaction to Ivalon in the human nose. A marked difference was noted in the degree and type of reaction. The hypothesis was made that this difference was due in part to the high degree of tissue immunity (or resistance) to Ivalon inherent in nasal tissue as contrasted to that present in heart tissue.

ACKNOWLEDGMENTS.

I take this opportunity to acknowledge my indebtedness and to express my gratitude to Dr. Edward E. Siegler, Director of Laboratories and to Dr. John J. Kralik, Head of the Department of Cardiovascular Surgery, both of Marymount Hospital, Garfield Heights, Ohio, for the splendid cooperation that they gave me in the preparation of this paper.

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9119 Miles Avenue.

AMERICAN HEARING SOCIETY MOVES.

National headquarters of the American Hearing Society were moved to 919 18th St., N. W., Washington 6, D. C., during the Christmas holidays. The agency occupies first floor space in an eight-story building which is being remodeled and converted from apartments to offices.

LOW GRADE CANCER OF THE ETHMOID SINUS.*

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Cancer that involves the paranasal sinuses is one of the most tragic diseases that we, as otolaryngologists, encounter. At least 60 per cent of the patients that have this condition do not survive five years, in spite of the best available treatment. Those who do survive usually have significant deformities and annoyances.

Resection of the upper jaw for this condition has been carried out for more than 125 years. When Blandin[1] reported his case in 1834, he stated that several other authentic cases of removal of the superior maxilla had been recorded.

At the beginning of this Century interest developed in the destruction of these tumors with the actual cautery. Success with this method led to the use of electrocoagulation with one of the machines that destroys tissue by extreme dehydration. The results reported following the use of this method by such men as New,[2] Ohngren,[3] Schall[4] and others led to considerable optimism in dealing with this problem, since up to 40 per cent of the patients treated in this manner survived five years.

Several of these reports were based on quite a large number of patients.

In more recent years enthusiasm for a return to conventional cold instrument surgery in resection of these tumors has developed in some quarters. This seems to have followed the advances that have been made in the handling of surgical problems in general, such as in anesthesia, blood replacement, antibiotics, skin grafting and teamwork.

Cold instrument resection of the upper jaw can now be

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carried out with a low degree of postoperative mortality. It is an excellent operation for the teaching of surgical technique, since it follows certain known anatomical routes and can be described, demonstrated and illustrated; however, available statistics show the best results are obtained in those cases which have been treated by a plan which includes electrocoagulation and, in certain cases, irradiation.

In the past 15 years approval of the use of electrocoagulation has been expressed by Cranmer,[5] Poulsen,[6] Larsson and Martensson,[7] Hara,[8] Erich[9] and others. Hara[8] records the fact that in one hospital where 30 cases of cancer of the sinuses had been treated, the only two that survived five years had been treated by electrocoagulation.

Time does not permit an adequate discussion of this phase of the subject. In 1952 the speaker reviewed the literature thoroughly, and reported to this Society the relative results following the various methods and combinations of methods of treatment of cancer of the sinuses.[10] A review of the recent literature confirms the impression that there is a distinct advantage in the use of electrocoagulation in experienced hands.

About 80 per cent of cancers which involve the paranasal sinuses originate in the maxillary sinus. It is likely that some of those that originate in the ethmoid sinus spread to the maxillary so soon that the exact point of origin cannot be determined. The treatment of cancer of the ethmoid sinus in a large per cent of such cases is simply an extension of the treatment that is carried out for that of the maxillary; however, in some cases the tumor has originated in the ethmoid sinuses with little question, although extension into the other sinuses may have taken place to a certain degree. Larssen and Martensson[7] found 19, or 5.6 per cent of 334 cases arising from the ethmoid. Hara[8] found that in 5 or 5.5 per cent of 92 cases the ethmoid air caps alone were involved. Havens and Thornell[11] reported four cases of cancer originating in the ethmoid sinus. Harrison[12] reported two cases of lymphosarcoma of the ethmoid. We have treated two cases of relatively high grade cancer originating in the ethmoid sinus. The eye was sacrificed in both cases and electro-

coagulation carried out through the fronto-ethmoid incision.[10] Radium element was placed in the cavity immediately. Supplemental Roentgen therapy was given both cases at the suggestion of the Roentgenologist. These patients are without local recurrence nine and twelve years respectively. One patient developed cervical metastasis in the opposite side and three months later on the side of the sinus tumor. Radical neck dissection was carried out on each side at separate times and he is without recurrence six years since the last operation.

Single cases of cancer of the ethmoid have been reported by Munson and Munson,[13] Harrison,[12] Schall,[14] Hibbs and Telch,[15] Tolczynsky,[16] Harper[17] and High.[18]

The type of cancer involving the sinuses is squamous cell carcinoma (epidermoid) in a high per cent of cases. Sarcoma is much less common and adenocarcinoma relatively rare. Further pathological classification may be confusing, due to a certain variation in terminology and is consequently beyond the realm of this paper; however, since adenocarcinoma of the cylindroma type was present in one of the case reports to follow, something of its characteristics and its relative frequency of occurrence is thought to be of interest.

This tumor may be called adenocarcinoma, adeno-cystic carcinoma, adenoid cystic carcinoma, adenoid cystic basal cell carcinoma, adenoid cystic epithelioma, basal cell carcinoma with hyaline stroma, adenomyoepithelioma, adenocarcinoma of cylindroma type, cellular mixed tumor, basaloma and cylindroma.

In 1859 Billroth[19] described a cylindroma of the parotid gland and referred to an earlier case of cylindroma of the orbit. He is generally given credit for the first description of the tumor.

Histologically the cylindroma is characterized by the arrangement of small darkly staining cells with relatively little cytoplasm, closely contiguous in anastomosing cords between which are rounded cellular areas which may contain mucus, hyaline or muco-hyaline material.

Beck and Guttman[20] used the term basaloma for this

tumor and found but 37 cases involving the respiratory passages and their adnexa in the literature and added three cases of their own. Although the maxillary antrum was involved in over half of them there was no case in which the ethmoid alone was involved.

One of the most important characteristics of the cylindroma from a practical standpoint is the lack of correlation between the histopathology and clinical behavior. While the microscopic picture is one of a very low grade of malignancy, the tendency to local recurrence and metastasis is often marked. Attention has been brought to this fact by Pollock.[21]

Frequency of Occurrence of Adenocarcinoma and Cylindroma in Cancer of the Paranasal Sinuses.

Author	Cancer Cases	Adeno-Carcinoma	Per cent	Cylindroma	Per cent
Larsson and Martensson	334	11	3.3	34	10.2
Schall	219	22	10	1	.4
Geschickter	139	15	10.8	15—Cystic Basal Cell Ca.	10.8
Watson	121	11	9	0	0
Hara	92	10	10.8	0	0
Cranmer	91	5	5.5	2	2.2
New	91	6	6.6	0	0
Beatty	37	3	8	0	0
Poulsen	19	2	10.5	0	0
Haas	23	0	0	1	4.3
McKenzie	10	0	0	1	10
Totals	1176	85 (7.2%)		54 (4.6%	

Fig. 1.

A combined total of 1176 cases of cancer of the sinuses reported in eleven articles included 85 which were classified as adenocarcinoma, an incidence of 7.2 per cent. In addition there were 54 cases classified as cylindroma, an incidence of 4.6 per cent. This group included 15 cases called cystic basal cell carcinoma rather than cylindroma (see Fig. 1).

While the occurrence of cases classified as adenocarcinoma was rather consistent in the various reports, that of cylindroma was not. Five authors reported no cylindroma while two reported an incidence of over 10 per cent. This may be largely due to the variation in terminology.

Schall[22] feels that the surgical approach to tumors of the sinuses should depend on the point of maximum involvement. He states that if exophthalmos exists and if it is questionable whether the orbit has been involved, the ideal approach is through the orbit by an exenteration of the orbital contents.

In 1944 Havens and Thornell[11] thoroughly reviewed the literature dealing with the development and technique of the

Fig. 2a. Squamous Pappilloma, X21.

fronto-ethmoid incision and reported its use on eight cases of carcinoma of the nasal cavity and paranasal sinuses.

As a rule the degree of malignancy of cancer of the sinuses requires the most active and extensive treatment that one feels is consistent with safety. There are times, however, when the tumor is thought to be of sufficiently low grade to justify some conservatism in its treatment. This fact, combined with the unusual occurrence and characteristics of the following two cases, is the reason for reporting them:

Case 1.—A white man, 35 years of age, was seen by a well-qualified Detroit otolaryngologist in 1939. He complained of inability to breathe

through the left side of his nose and intermittent attacks of pain over the left frontal and maxillary sinus region. There was history of having had bilateral iridectomies for glaucoma in 1932. There was no useful vision in the right eye. A left Caldwell-Luc operation was performed and multiple polyps removed from the left nasal cavity. A partial removal of the left middle turbinate was done also.

A well qualified Detroit pathologist made the following report: Squamous and columnar epithelium surmounting a loose areolar connective tissue. A few lymphocytes are infiltrating the stroma. Other sections are covered by stratified columnar epithelium. The stroma is edematous and myxomatous and infiltrated by lymphocytes.

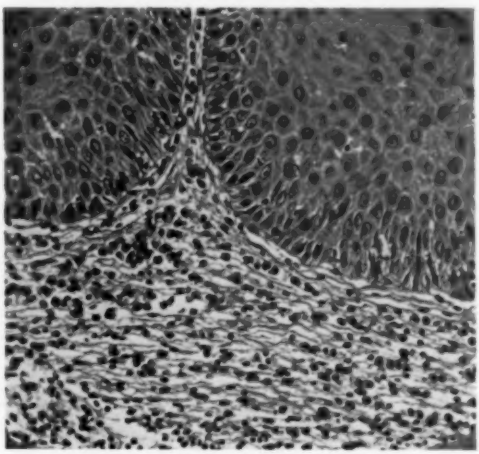

Fig. 2b. Squamous Papilloma, X185.

Diagnosis: Infected nasal polyps.

Polyps were removed again in 1942, 1944 and 1945.

The pathological report in 1945 was: Chronically infected nasal polyps with excessive hyperplastic surface epithelium. A foot-note stated: "It is my opinion this polyp would become squamous cell carcinoma if allowed to develop further. It is on the border of malignancy at this time."

In 1946, one year later, microscopic examination of removed tissue was reported in much the same way.

At this time the patient was referred to an excellent out-of-state medical center, where the chief pathologist and chief otolaryngologist and an associate agreed that the tumor in the ethmoid region would probably remain benign, and more radical surgery was considered optional. It was

recommended that he be seen every six months for several years. This was done.

I saw him first on February 19, 1950, at which time there was a polyp 6 cm. in diameter in the left middle meatus. He returned to the out-of-state medical center, where a submucous resection was carried out with removal of polyps from the left ethmoid region. A left naso-antral window was made also. This was presumably an enlargement of the original antrum window put in at the time of the Caldwell-Luc.

I removed tissue from the ethmoid region in May, June and July of 1950.

In 1952 I removed more tumor and sent him to the out-of-state medical

Fig. 3a. Squamous Cell Carcinoma, X21.

center for re-evaluation. After examining the recently removed tissue it was decided that the tumor was still benign and surgery optional.

I removed more of the tissue in 1952 and again in 1953, at which time the Detroit pathologist made essentially the same report as he had previously: Squamous epithelial and adenomatous polyps of nose. Squamous type tend to recur and sometimes become malignant (see Figs. 2a and 2b).

Nine months later I removed further tissue, and the diagnosis by a local pathologist was squamous cell carcinoma. A slide was sent to the out-of-state medical center, where the diagnosis of carcinoma was confirmed, and it was termed pseudo-papillary squamous cell epithelioma. It was considered not infiltrative and not remarkably high-grade. There were other areas which showed benign papillomatous structure (see Figs. 3a and 3b).

Roentgenograms at this time showed cloudiness of the left ethmoid sinus with involvement of the left frontal and maxillary (see Fig. 4).

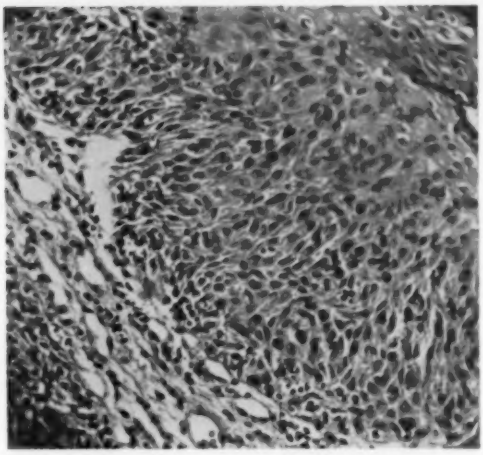

Fig. 3b. Squamous Cell Carcinoma, X185.

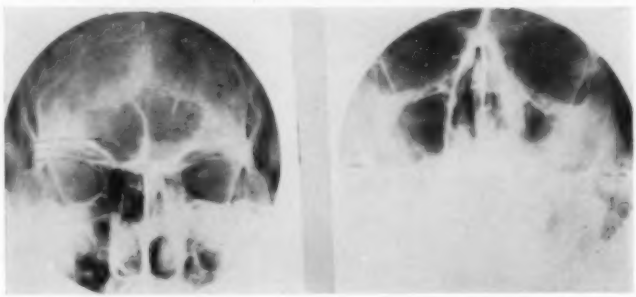

Fig. 4. Roentgenograms of Sinuses showing cloudiness of left ethmoid, frontal and maxillary sinuses.

There seems little question that the transformation of nasal polyps to carcinoma occurred in this case. Tolczynsky[16] states that such an occurrence is extremely rare, and this fact is no doubt responsible for the long delay in carrying out more radical surgery.

Schall[14] reported the case history of a woman who had had polyps removed every year for about 10 years with no micro-

scopic examination. When this was done it revealed epidermoid carcinoma of the ethmoid labyrinth.

On September 14, 1958, the following operation was carried out by the author. Under Pentothal Sodium anesthesia an incision was made extending along the left side of the nose upward medial to the inner canthus and along the lower border of the inner third of the eyebrow. The skin and periosteum was elevated anteriorly and then posteriorly as far as the anterior ethmoid artery, which was ligated and then cut on the nasal side.

The anterior ethmoid cells were entered through the lacrimal fossa. These cells contained cancer. A complete ethmoidectomy was carried

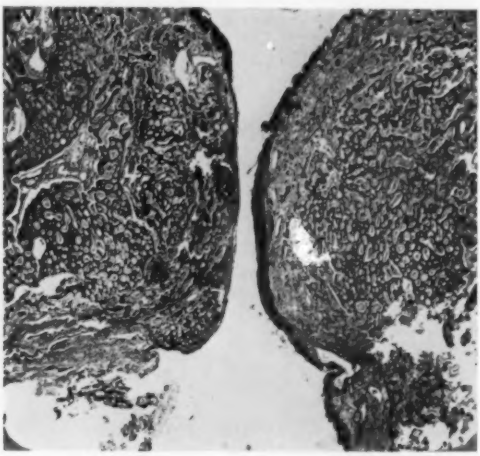

Fig. 5a. Cylindroma, X21.

out. The floor of the frontal sinus was then removed as was the anterior wall of the sphenoid sinus. The edematous and swollen mucous membrane was removed completely and thoroughly from these sinuses. The left maxillary antrum was entered from the ethmoid region and the mucous membrane removed from it. Thorough electrocoagulation of the soft tissue surrounding the operative field was carried out but it was felt unneccessary to electrocoagulate the remaining bone.

The wound was closed with heavy Dermalon retention sutures and fine silk skin sutures.

A 26-F rubber tube was passed into the frontal sinus through the nose and left in place for ten days.

Microscopic examination of all the tissue removed at operation indicated that the tumor extended into the maxillary and sphenoid sinus to a limited degree.

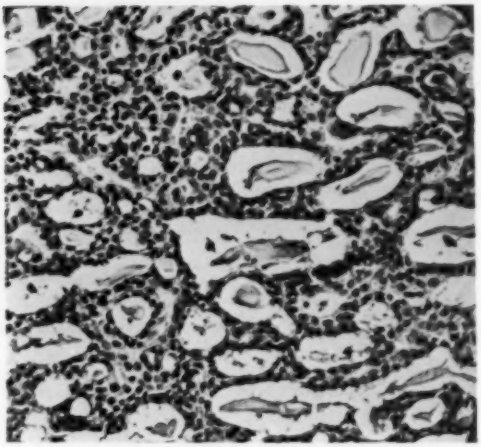

Fig. 5b. Cylindroma. X185.

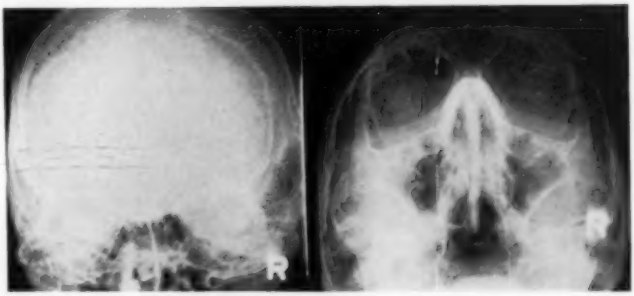

Fig. 6. Normal Roentgenograms of the Sinuses.

The patient has been symptom free for four and one-third years. Biopsies of small tags removed from the ethmoid region on several occasions have been reported squamous papilloma, but there has been no significant recurrence of the tumor. He is followed closely, however, and the possibility of having to reoperate upon him in the future is recognized.

Case 2. A white woman, age 55, was first seen on May 26, 1958. She stated that a year previously she had noticed a swelling developing between the bridge of the nose and the inner canthus of the right eye. This had grown gradually. For a year she had had pain in and around the right eye radiating to the right half of her head and down to her neck. There had been recent recurrent bleeding from the right side of the nose.

On examination there was a firm subcutaneous tumor about 1 cm. in diameter just medial to the inner canthus of the right eye and extending up to the region of the floor of the frontal sinus. It was not movable and not attached to the skin.

On examination of the right nasal cavity there was found a polypoid tumor 3 to 4 cm. long and apparently arising in the ethmoid region. Biopsy of this was reported adenoid cystic carcinoma (cylindroma) (see Figs. 5a and 5b).

It was considered a low grade of malignancy and of slow growth but prone to local recurrence and distant metastasis, particularly to cervical lymph nodes.

Roentgenograms of the paranasal sinuses were entirely normal (see Fig. 6).

General physical examination and laboratory studies including an electrocardiogram revealed no significant abnormalities.

Roentgenograms of the chest showed pulmonary emphysema and fibrosis. Gall-bladder studies were normal.

On June 5, 1958, through a fronto-ethmoid approach the tumor, 1½ to 2 cm. in diameter, was encountered just under the skin and subcutaneous tissue having eroded through the bone. It extended through the ethmoid sinus region and into the nasal cavity, being 4 to 5 cm. long and free of attachment except in the ethmoid region. After removal of the tumor the surrounding area was electrocoagulated thoroughly. This included the right side of the nasal septum and destroyed most of the right nasal bone and the nasal process of the superior maxilla on the right side. Since the frontal duct was involved, the floor of the frontal sinus was removed entirely so that the orbital contents could obliterate it as completely as possible. The partition between the frontal sinuses was removed so that drainage could pass to the left side if necessary.

A number 26-F rubber catheter was passed into the frontal sinus through the nose and the wound was closed with Dermalon and fine silk sutures. The patient was dismissed from the hospital on the seventh postoperative day, and the rubber drainage tube removed at that time.

Five months later there is no evidence of recurrence.

CONCLUSION.

Two cases of primary cancer of the ethmoid sinus of relatively low grade have been reported in some detail. The first case is most unusual, because there appears little doubt that squamous cell carcinoma developed in benign polypoid tumors that had repeatedly recurred over a period of 15 years. The rarity of this occurrence is no doubt responsible for the long period of time before more radical surgery was carried out. Extensive electrocoagulation and heavy irradiation was thought unnecessary because of the low degree of malignancy of the tumor and unwise because of blindness in the opposite eye.

The second case is interesting chiefly from the standpoint

of the relatively rare cylindroma originating in an ethmoid sinus.

Advantage has been taken of the opportunity to discuss briefly cancer of the paranasal sinuses in general.

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The Eighth Congress of the Pan-Pacific Surgical Association will be held in Honolulu, Hawaii, September 28 through October 5, in 1960.

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The clinical problems of benign lesions of the lower end of the esophagus are basically anatomic problems, and the controversy started in 1719 by Helvetius'[6] description of a morphologic sphincter at the cardioesophageal junction still exists. Mosher[11,12] at one time asserted that the cardiac sphincter was definite and at a later date stated that it did not exist.

A great deal of information on gastroesophageal regurgitation and hiatal hernia has accumulated in the literature in recent years and pressure readings at the lower end of the esophagus, as performed by Olsen[13] and others[2,4,7] have helped to determine the part played in gastroesophageal regurgitation by both intrinsic and extrinsic factors, causing elevation of intragastric pressures. Even though these manometric pressure studies have increased our knowledge, the exact mechanism of gastroesophageal sphincter function remains in dispute. The prevailing opinion today is that a physiologic sphincter is present at the esophagogastric junction, and while it does not have the anatomic features of a true muscular sphincter it acts in such a way as to prevent reflux regurgitation of material from the stomach into the esophagus unaided by any pinch-cock action.

Lendrum[5] demonstrated that the distal esophageal epithelium terminates not in a straight line but in a jagged, irregular and angulated line. The constriction at the esophagogastric junction is formed by a thickening of the circular muscular fibers. There is no evidence to support the theory that the hiatus acts as a pinch-cock to close the cardiac end of the esophagus, for its grip on the esophagus is insufficient to prevent the normal sliding movement which occurs during respiration. Davis[5] demonstrated that the level of the esopha-

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gogastric constrictor in relation to the diaphragm was not fixed, and was within or above the hiatus during descent of the diaphragm in inspiration and fell below the hiatal level during expiration as the diaphragm rose. The esophagogastric junction is located immediately below the inferior esophageal sphincter and is continuous with the cardiac constrictor, which is formed by a continuation of the circular muscle fibers of the esophagus reinforced by eliptical muscle fibers and covered by thick mucosa. When viewed esophagoscopically the esophagogastric junction appears as a stellate or puckered orifice with a horizontal opening which remains closed except during the act of swallowing. This sphincter-like area has been found histologically to consist of a fold of mucosa with deep thickened muscular fibers which act similar to valves. The flap valve of mucosa at the exact point of junction of the esophagus and stomach offers no obstruction to the downward flow of material but is resistant to reflux, and pressure studies by Barrett[3] indicated that it operated over not more than a 5 mm. area. The efficiency of this mechanism is borne out clinically by the difficulty encountered in finding the cardiac opening into the esophagus when retrograde esophagoscopy is performed through a gastrostomy opening, and at times considerable pressure has to be exerted upon the mucosa at the esophagogastric junction in order to enter the esophagus from below. Allison[1] noted also that when the esophagus and stomach were removed from the body greater pressure was needed to drive fluid from the stomach into the esophagus than in the opposite direction. Even though no actual internal sphincter can be demonstrated there is an intrinsic mechanism contributing to continence at this level, and recent experimental work by Meiss and his associates[10] indicate that it lies within the esophagogastric junction.

The maintenance of a competent esophagogastric sphincter mechanism seems to be dependent upon: *1.* an intrinsic morphologic sphincteric factor as yet ill defined; *2.* the contraction of the circular muscle fibers of the esophagus and the oblique fibers of the stomach as they come together at an angle; *3.* the valve-like flap of mucous membrane in this area, and *4.* the diaphragmatic crus. The latter alone cannot in-

sure continence, and the size of the opening in the right crus of the diaphragm is so variable that an opening which is larger than normal does not always indicate a hiatal hernia. Normally the mucosa of the esophagus is protected from the acid gastric juice by the secretion of mucus and saliva and by its squamous epithelial lining. Regurgitation of gastric secretion into the lower esophagus is present in some cases and can be produced by increased intragastric pressure and distention without an actual hiatal hernia being present. In 1926 Robins and Jankelson[14] observed, in 103 cases studied roentgenologically, that 4.6 per cent had cardioesophageal relaxation in a variety of clinical lesions without any demonstrable esophageal disease.

TABLE I.

Diagnosis in 184 Patients with Symptoms of Dysphagia and Pain from Lower Esophageal Disease.

	Hiatal Hernia	Esophagogastric Incompetency
Roentgenography	142	2
	161	23
Esophagoscopy	19	21
Esophagitis	39	3

Gastroesophageal reflux with its resulting esophagitis is the cause of the most disabling symptoms of hiatal hernia, and the size of the hernia is no indication of the nature of the reflux or the amount of ulceration present. The severity of the symptoms does not always agree with the extent of the esophagitis, and the pain at times is not proportionate to the visible inflammation.

In a group of 184 patients (see Table I) seen during the past five years with symptoms of esophagitis, a definite hiatal hernia was demonstrated on roentgenographic study in 142 cases. In the remaining 42 cases with a normal esophagogram a hiatal hernia was found on esophagoscopy in 19 cases, and an incompetent sphincter without hiatal hernia was present in the other 23 cases. In approximately one-fourth of the 161

cases with hiatal hernia esophagitis, with or without ulceration, was observed esophagoscopically, but only three of the 23 cases of esophagogastric sphincter incompetency exhibited actual ulceration.

In all of the cases observed the symptoms were those of esophagitis. Dysphagia of some degree usually occurred on eating solid foods, and lodgment of food not infrequently resulted so that the patients found it necessary to take large quantities of fluids with meals. Regurgitation was prone to occur after food intake and often interrupted the course of a meal. Bending forward after eating produced regurgitation into the mouth, and the material or sour fluid regurgitated had to be swallowed again. This usually left the throat feeling dry or burny. Sometimes patients noticed lodgment of food at the lower end of the sternum, accompanied by pressure sensations that were relieved as the bolus passed into the stomach.

The most severe symptom was that of pain either precordial, epigastric, in the back, or radiating upward into the shoulder, or downward into the abdomen. The distress varied from a few minutes to hours with a corresponding inconstancy in the time interval, but as the esophagitis became more marked the pain became more constant and severe and increased on stooping or bending. It commonly awakened patients at night, especially if they were sleeping on their backs, and relief was obtained by sitting upright or taking water, milk or alkalies. The presence of gas and marked belching was also observed. These symptoms are also similar to those of peptic ulcer, a condition from which esophagitis must be distinguished.

The diagnosis of hiatal hernia can be made both roentgenologically and esophagoscopically, and it is now generally agreed that patients with symptoms of esophagitis should have an esophagoscopic examination, even though the Roentgen findings are negative. With a positive Roentgen finding of hiatal hernia esophagoscopy establishes the presence and extent of the esophagitis as well as confirms the diagnosis.

In the 23 cases of esophagogastric sphincter incompetency the symptoms were explained by the finding on esophago-

Fig. 1. Roentgenogram of a patient with sphincter incompetency without hiatal hernia. Esophagoscopy revealed a relaxed and incompetent esophagogastric junction in its normal location, absence of the normal angle of entry of the esophagus into the stomach and extensive ulcerative esophagitis.

scopic examination of an incompetent esophagogastric junction with loss of the normal angle of entry of the esophagus into the stomach (see Fig. 1). In these cases no actual herniation was observed, yet the symptoms were due to the incompetent hiatal constriction. Additional esophageal find-

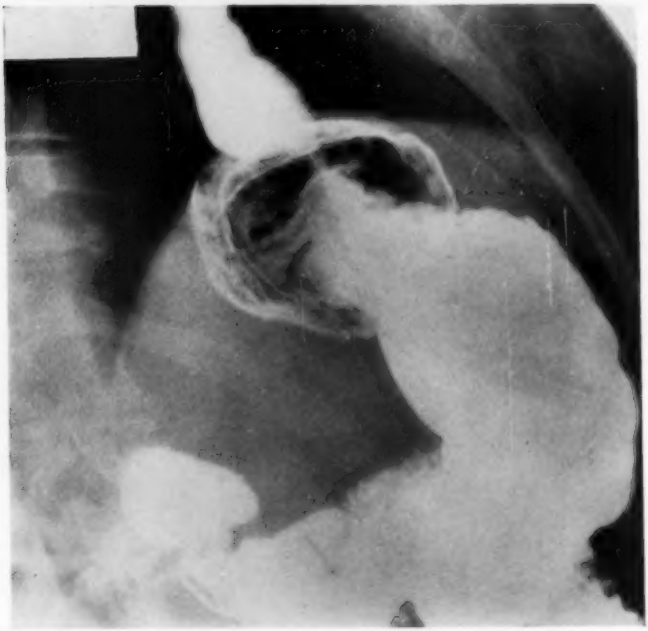

Fig. 2. Postoperative roentgenogram of a patient after hiatal hernia repair in whom the esophagus was too short to allow displacement and establishment of an angle at the esophagogastric junction. Even though the stomach remained below the diaphragm the persistence of the direct entry of the esophagus into the stomach permitted incompetency and esophagitis.

ings were thickening of the mucous membrane with some redness and redundancy of the folds at the hiatal level. The mucosa often bled easily, and occasionally small ulcerations were present. Between the ulcerations which, varied from red to yellow, the mucous membrane was thickened and swollen. Granulation tissue when present was usually flat and did not project into the lumen. The esophagus was found to enter the stomach directly in a straight line rather than at an angle to the left and superiorly as is normally observed. In one case extensive ulceration existed, which subsequently healed with narrowing and stenosis consisting of a funnel-like constriction, but no visible scarring similar to that seen in

acquired strictures developed. In another case operated upon for repair of a hiatal hernia, the esophagus was too short to allow mobilization and establishment of an angle at the cardioesophageal junction, and although the stomach was placed below the diaphragm, incompetency persisted because of the vertical entry of the esophagus into the stomach (see Fig. 2). While the use of inflation in the lower esophageal segment is useful in visualizing a hiatal hernia with marked redundancy of the mucosal folds, ballooning of the hiatal area with air was rarely needed to demonstrate esophagogastric incompetency.

It has been my feeling that relaxation and incompetency is the first step in the development of a hiatal hernia, and these cases eventually progress to actual hernia formation. Since relief has thus far been obtained in this series by medical treatment, there has been no indication for further esophagoscopic examination. On the other hand, esophagogastric sphincter incompetency may simply be a manifestation of a reducible hiatal hernia and the incompetency results from the hernia. This latter hypothesis was not confirmed in the one case in which surgical exploration was performed, for no hernia could be demonstrated at operation. Wyllie and Field[16] reported six infants, age 1 month to 18 months, with intermittent esophageal regurgitation and hematemesis in which esophagoscopy revealed absence of the constriction at the esophagogastric junction, and incompetency of the sphincter but no actual herniation. In another case these identical findings were present in an infant who developed a definite hiatal hernia four weeks later as demonstrated esophagoscopically.

In recent years sporadic cases of esophagogastric sphincter incompetency have been treated surgically with reported relief of symptoms. Lortat-Jacob[9] sewed the greater curvature of the stomach to the lower esophagus, and Stensrud[15] performed a similar operation for incompetent cardia by suturing the fundus of the stomach to the left dome of the diaphragm.

Uncertainty remains as to the permanent results of medical treatment, but all of the patients in this group with esophago-

gastric incompetency improved under medical management. Further study is needed to determine the subsequent development of a hiatal hernia. Proper diet with thorough mastication of food afforded the most relief and bland low residue diets were used. Aggravation of symptoms usually followed a heavy or large meal, and avoidance of large quantities of food and swallowing small amounts at a time were helpful. Liquids taken during the course of a meal aided in washing down the food, particularly if there was a tendency for lodgment at the esophagogastric junction. Any constriction or increased intra-abdominal pressure produced an increase in the symptoms and intensified them. Reclining after eating was detrimental, and relief was obtained if the patient sat upright. Sleeping in a semi-recumbent position was practiced routinely, and alleviated much of the night pain. Careful attention to the diet usually sufficed so that medication was rarely indicated, but if so, alkalies and aluminum compounds were employed.

SUMMARY.

A mechanism exists at the esophagogastric junction, although its true nature has not been accurately established, which acts as a morphologic sphincter and prevents retrograde flow of gastric secretion. When incompetency of the sphincteric action develops symptoms indistinguishable from those of esophagitis occur.

In a series of 184 cases with esophagoscopically demonstrable esophagogastric sphincter incompetency, the cause was due to hiatal hernia in 87.5 per cent, and associated esophagitis was present in one-fourth. In the remaining 12.5 per cent, incompetency of the esophagogastric sphincteric mechanism was the only finding, yet esophagitis was observed esophagoscopically in only three patients. Medical treatment in the latter group has produced relief of symptoms.

Esophagogastric sphincter incompetency is the initial step in the development of hiatal hernia in some cases, but further observation is necessary to prove this relationship in other cases.

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USE OF ACRYLIC MATERIALS IN LARYNGOLOGY.*†

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The problem of chronic stenosis of the larynx and upper trachea is a familiar one to laryngologists. Although there has been a lessened incidence of the post-diphtheritic type and that due to faulty (high) tracheostomy, with subsequent perichondritis of the cricoid, this has been partially balanced by an apparent increase in the number of cases of traumatic origin. Among the less important causes are acute non-specific infections, tuberculosis, syphilis, cicatricial narrowing following operations on the larynx, and perichondritis following irradiation or intubation of the stomach ("feeding-tube stenosis").

The important and well-known items in pathogenesis are annular fibrosis and overgrowth of cartilage, the usual sequelae of perichondritis, and in some cases loss of support due to necrosis of cartilage. An added factor, important in children and emphasized many years ago by Jackson, is failure of normal growth of the portion of the airway which is not functioning.

Simple direct laryngoscopic dilatation has been used with success in many cases, particularly in childhood, where the stenosis is not of marked degree and where the tracheostomy has been performed at the proper level. For some time, however, it has been recognized that more rapid progress can be made by the use of indwelling apparatus in increasing sizes. Plastic procedures, with or without skin graft, have been used in instances of excessive overgrowth of fibrous tissue or cartilage, as well as in cases where support is deficient because of loss of cartilage. In nearly all cases of marked

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stenosis, however, the use of indwelling molds will constitute an essential part of the treatment.

In the past, molds of several types have been used. Examples of these are the hard-rubber O'Dwyer tube, rubber core-molds of shape similar to the O'Dwyer tube and sections of ordinary urethral catheter, the latter having the advantage of being easily adapted to the individual case. The rounded end rubber dilator of Jackson has been useful in the treatment of stenosis of the upper trachea.

The most troublesome problem in the use of indwelling rubber apparatus has been the degree of inflammatory reaction induced. Despite frequent changing, persistent mucosal ulceration with granulation tissue formation is frequent, and even reactivation of perichondritis may be observed. Ideally, the material used for core-molding should be free of irritation, non-porous, easily shaped and tolerated with comfort for periods of weeks or months.

Acrylic materials, which fulfill these criteria have found application in several fields. In addition to dental and maxillofacial uses, acrylics have been employed in arthroplasty, spine stabilization and cranioplasty, for plombage in pulmonary tuberculosis, as experimental aortic valves, as orbital implants and as corneal lenses. In 1945 Erich[4] described the use of veronite (a dental acrylic material) in the treatment of chronic laryngeal stenosis.

The Acrylics.

The acrylics (acrylic resins) constitute one class of plastics, a large group of organic substances, wholly or in part synthetic, which can be molded into solid articles retaining their shape indefinitely at ordinary temperatures. The components of plastics have resinous characteristics, *i.e.*, they lack a visible crystalline structure because of the size and complexity of their molecular structure. Resinification may occur by condensation (a chemical reaction which eliminates H_2O or some other simple compound) or by polymerization, in which high-molecular compounds are formed by consecutive joining of unsaturated molecules.

Acrylic resins are examples of polymers of acrylic acid and its derivatives. The acrylates were first produced commercially in the late 1920s; in 1936 methyl methacrylate resin was introduced as "Plexiglas" by the Rohm & Haas Company, and as "Lucite" by E. I. duPont de Nemours and Company.

Methyl methacrylate, the acrylic which has been most used in medical work, is colorless, transparent, and may be highly polished, with a surface comparable in hardness to copper or brass. It is not affected by aqueous solutions of inorganic salts or by dilute acids or alkalies, but alcohol may cause softening and "crazing." The optimum temperature for molding is 245° to 310° F.; at higher temperatures the formation of small bubbles will destroy the smoothness of the surface. Holes may be drilled with ordinary twist drills, with care to avoid over-heating from friction. Particularly important is the high biologic tolerance for the material, which elicits a minimum of inflammatory response in living tissues.

Acrylic Molds.

Solid acrylic molds are usually prepared in the dental laboratory by casting, in the same manner as dental plates, from a previously prepared model formed by the use of dental modeling compound and base plate wax. Chemically, the process consists of introducing a "syrup" of partially polymerized monomer into the plastic mold; solidification occurs as polymerization becomes complete. Special "plasticizers" may be used as agents to complete the polymerization.

Frequently a rubber mold which has been previously worn may be used as a starting point in preparation of the model which is sent to the dental laboratory. As suggested by Robb,[12] barium sulfate powder is incorporated in the mixture at the time of casting if a radio-opaque mold is desired; the opacity of methyl methacrylate alone is about the same as that of soft tissue.

Hollow acrylic molds may be made by drilling a solid mold lengthwise, but are prepared more quickly and more conveniently by heat-shaping of acrylic tubing, which is supplied in several sizes with a wall thickness of one-sixteenth inch. Heat-shaping may be accomplished by the same general

methods used in shaping glass, with due precaution to avoid overheating. An ordinary alcohol lamp with a small flame serves well for this purpose. Acrylic tubing may be cut with a jeweler's saw or a small hack-saw, and the ends smoothed with the finest grade of sand-paper, followed by "crocus cloth" and a final buffing with pumice.

Core-Molding.

The objectives of core-molding may be: *1.* to prevent chronic stenosis (a) in cases of recent trauma with fracture of laryngeal cartilages, rupture of tracheal wall or dislocation of arytenoids, or (b) following operation, if large portions of cartilage have been removed, as in the more extensive types of partial laryngectomy,[6,7,11] in laryngofissure for extensive papillomatosis,[15] and in arytenoidectomy for bilateral paralysis[13]; *2.* To accomplish and maintain dilatation in cases where chronic stenosis has already occurred. Relief of stenosis may be achieved by plastic procedures with excision of fibrous tissue and cartilage, but stenosis will recur unless molds are used to maintain the lumen until the tendency for contracture has diminished (several weeks or months). In many cases, the use of molds alone, without preliminary operation except for tracheostomy, will be successful.

INJURIES OF THE LARYNX AND TRACHEA.

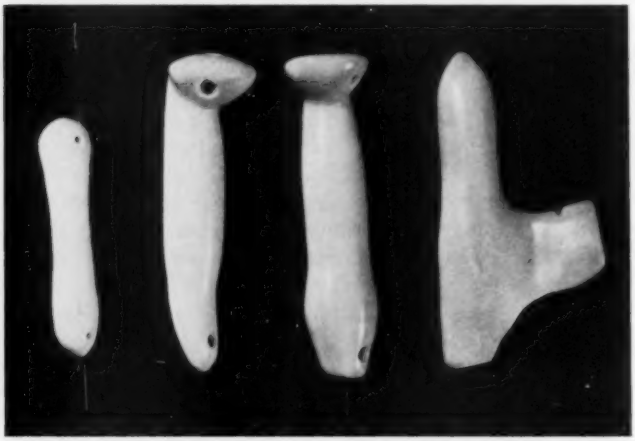
The management of these cases, which are encountered with increasing frequency, has recently been comprehensively reviewed by Holinger and Johnston.[8] In cases of recent trauma, an acrylic mold corresponding in size to the injured larynx should be inserted as promptly as possible, preferably at the time of tracheostomy, and left until healing is complete. The mold will function as an internal splint, aiding in restoring displaced fragments of cartilage to their normal position. Even though some degree of perichondritis may occur as a result of the injury, this will not be increased or unduly perpetuated by the presence of the mold if the proper size and shape are chosen. Systemic antimicrobial treatment, preferably based on cultures and sensitivities, may be used.

Examples of molds used for this purpose are shown in Fig.

1-b and Fig. 1-c. Incised wounds (windshield, attempted suicide, etc.) involving the laryngeal cartilages are treated in a similar fashion if fragmentation of cartilage has occurred.

PREVENTION OF POSTOPERATIVE STENOSIS.

In extensive types of partial laryngectomy, with or without skin graft, a foam-rubber mold covered with latex is inserted at the time of operation and left for about ten days, usually



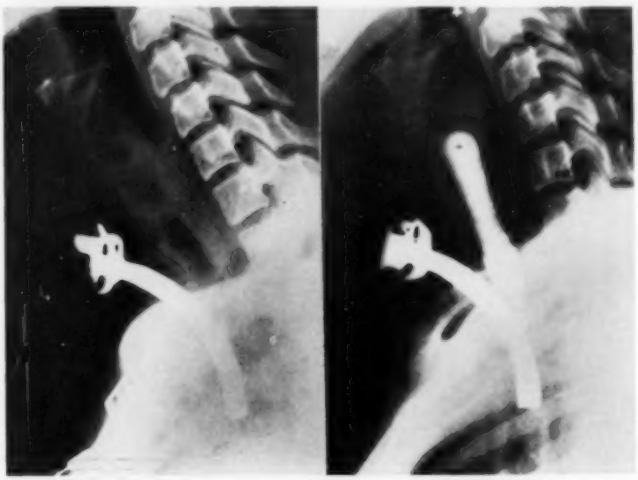
a. b. c. d.

Fig. 1. Solid acrylic molds. (a) Was used in the case illustrated in Fig. 2 (shaped to be self-retaining). (b) and (c) have been used in treatment of both recent traumatic stenosis and chronic stenosis. (d), shaped for introduction through an enlarged tracheal fistula, was used in the case illustrated in Fig. 3.

being anchored with two through-and-through heavy sutures of stainless steel wire. If progressive narrowing of the airway should occur thereafter, an acrylic mold should be inserted before the lumen becomes inadequate and left until epithelialization is complete and the tendency for further contracture diminished. Figi[6] has advocated the routine use of an acrylic mold in such cases. An acrylic mold is routinely inserted for two or three days following intralaryngeal arytenoidectomy according to the technique of Thornell.[13]

CHRONIC STENOSIS OF LARYNX AND UPPER TRACHEA.

Initially, the degree and extent of the stenosis must, of course, be evaluated by indirect and direct laryngoscopy, occasionally by "retrograde" laryngoscopy, and usually by Roentgen study including lateral films for best demonstration of the residual airway; planigrams are at times informative



a. b.

Fig. 2. (a) Nearly complete atresia of upper trachea in a girl of 12 years, the result of injury (fall on sharp edge of tin can) 6 years before. (b) Solid acrylic mold (28 f.) introduced after progressive dilatation with indwelling rubber molds (8 f. to 28 f.). The acrylic mold was removed after 6 weeks, with little evidence of subsequent contracture, and decannulation was accomplished after a test period of ten weeks. No further treatment required.

and films [illegible] following instillation of iodized oil may be helpful. Unless the stenosis is of such degree as to require a plastic operation, treatment should begin with increasing sizes of rubber molds (usually sections of catheter) or polyethylene tubing, changed at intervals of four to seven days to minimize inflammatory response. Attempts at vigorous dilatation and insertion of large molds initially may result in soft tissue and perichondrial inflammation sufficient to delay further treatment. Obviously, in cases where high tracheostomy

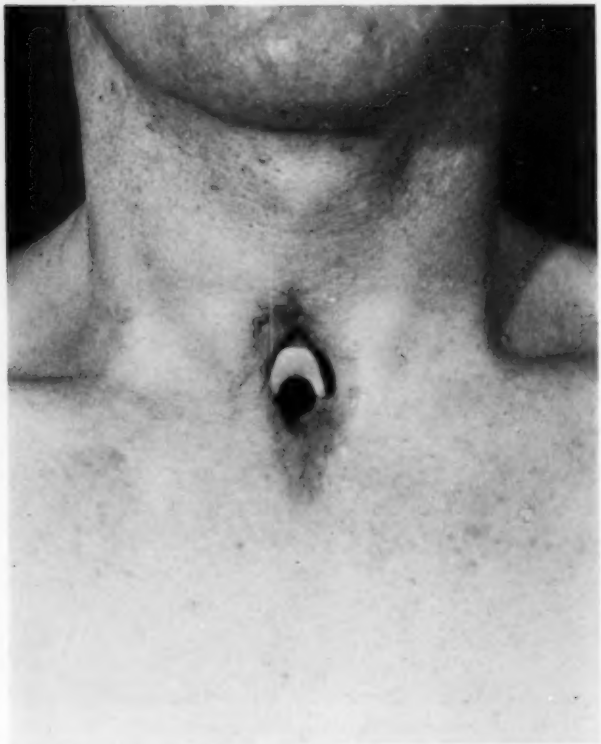


Fig. 3. Solid acrylic mold of special shape (see Fig. 1-d) in a case of atresia of upper trachea due to crushing injury (auto accident) four weeks before first seen. Preliminary treatment (six weeks) with increasing sizes of rubber molds (' f. to 34 f.) The mold shown was then worn continuously for twelve weeks; decannulation two months later. No recurrence of stenosis in past six years. Shape of mold allowed daily changing of tracheal cannula.

was an etiologic factor, a lower tracheostomy should be performed before treatment is started; in some cases, however, the upper fistula may be kept open and used to anchor the successive sizes of molds, and the subsequently used acrylic mold (see Fig. 5-b).

After accomplishing dilatation to the desired lumen size (average 32 to 36 fr. for the adult female or 36 to 40 fr. for

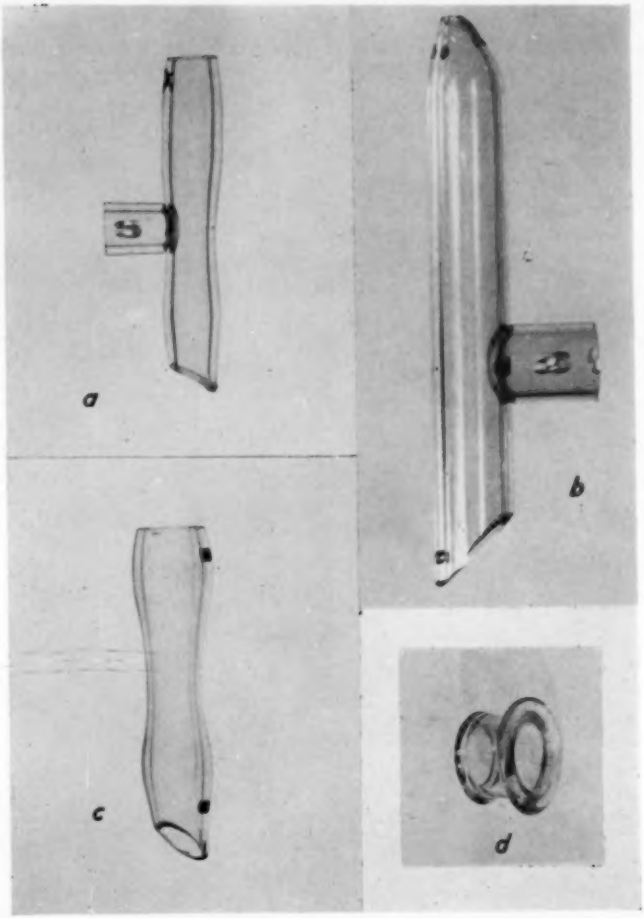
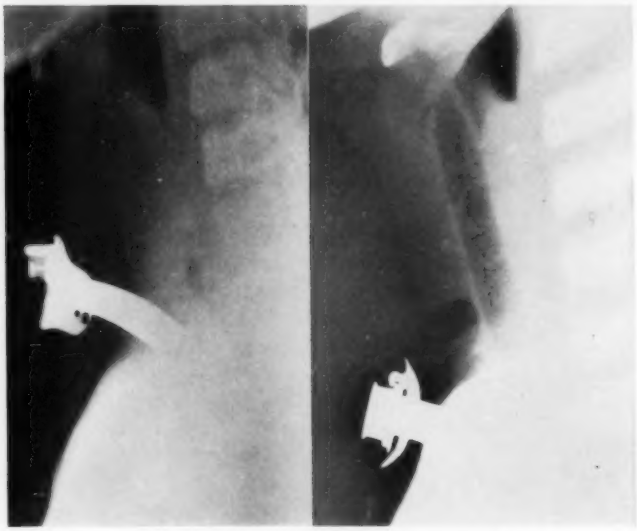


Fig. 4. Hollow acrylic molds, heat-shaped from acrylic tubing. (a) and (b) were used in a child and an adult, in which lower tracheostomy was required as a preliminary to treatment of chronic subglottic and upper tracheal stenosis. The separate short acrylic "post" lies in the fistula of previous tracheostomy, being anchored to the mold by heavy silk string. (c) is a hollow acrylic mold shaped to be self-retaining. (d) is an acrylic "button" (Moore) useful in preventing contracture of the tracheal stoma following laryngectomy.

the adult male), an acrylic mold of the same size is introduced. This may be done by, *1.* direct or suspension laryngoscopy, using a special forceps to grasp the upper end as the entering portion is advanced through the larynx, or *2.* drawing into place by axis-traction, using a heavy braided silk string loop passed through the hole in the entering end of the mold and manipulated through the tracheal fistula. A second loop of



a. b.

Fig. 5. (a) Marked glottic, subglottic and upper tracheal stenosis due to crushing injury (auto accident). (b) the hollow acrylic mold shown in Fig. 4-b is shown in place, having been introduced following progressive dilatation with rubber molds. The anchoring acrylic post lies in the uppermost (previous) tracheal fistula.

string through the upper end of the mold provides for counter-traction to maintain alignment. In the case of hollow molds, a third method may be used. First the mold and then a bronchoscope is introduced over the steel shaft of a previously inserted small bougie; the bronchoscope may then be used to push the mold into place. Less frequently, if there is a large anterior defect in the wall of the airway, the mold may be

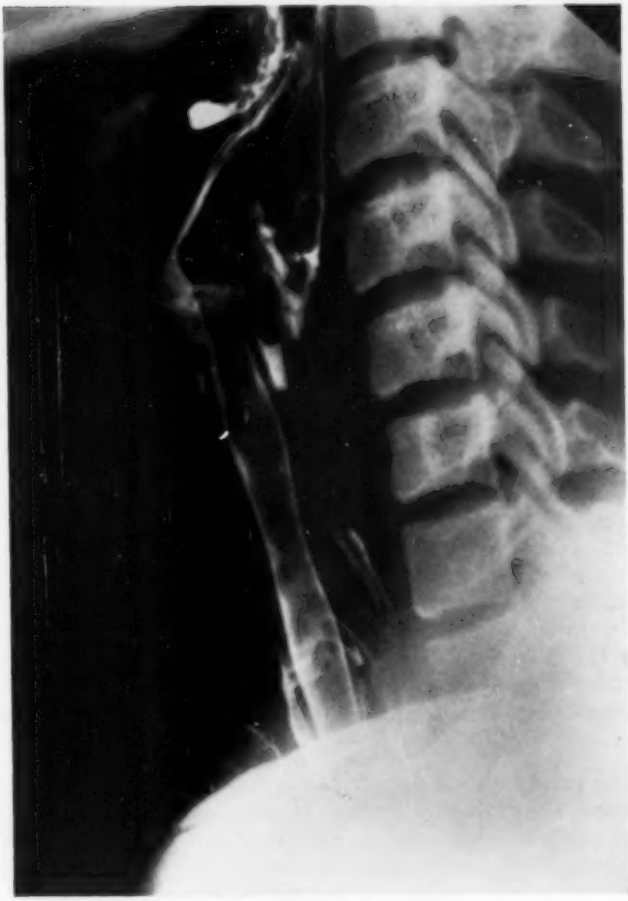


Fig. 6. Use of contrast material (Dionosil oily) to demonstrate relations of a hollow acrylic mold in a case of upper tracheal stenosis. Only the inner surface of the mold is coated. Position of upper end of mold just below glottic level is well shown. The outer end of the tracheostomic fistula is outlined. With cannula in place and occluded, patient has an excellent voice.

introduced from below. Laryngostomy is rarely required for introduction of a mold.

Molds may be kept in place, *1.* by shape, expanded portions at the upper and lower ends preventing movement in either direction (see Figs. 1-a, 2-b, 4-c, and 6); *2.* by anchoring by means of a loop of braided silk brought from the lower end of the mold through the tracheal fistula to a section of rubber or acrylic externally (tying to the cannula or its neck plate is not favored because this interferes with frequent changing of the cannula); or *3.* by use of a short "post" resting in the uppermost tracheal fistula and anchored against the surface of the mold by heavy silk ties (see Figs. 4-a, 4-b and 5-b). The latter method is useful in cases where previous "high" tracheostomy requires tracheostomy at a lower level. Specially shaped molds (see Figs. 1-d, 3) are inserted from below and have a protruding portion shaped to the convexity of the tracheal cannula. This prevents displacement and at the same time allows the patient to change the cannula daily.

The degree of reaction to presence of the mold may be determined by indirect or direct laryngoscopy. Increasing edema of the arytenoid eminences, increasing discomfort on swallowing, and tenderness, swelling or redness externally, indicate a disproportionate inflammatory response to a mold which is too large, or reactivation of perichondritis, in which case the mold should be removed and a smaller one introduced until resolution occurs. If no reaction is observed after two weeks, the mold will probably be tolerated for an indefinite period.

The usual interval for use of the acrylic mold is four to six months, although this depends to some extent on the degree of stenosis present initially. Hospitalization is not required after the first two or three weeks, but frequent out-patient observation is desirable. A test period of several weeks after removal of the mold, before decannulation, will determine whether there is any appreciable tendency for recurrence of stenosis.

Other Uses of Acrylic Materials.

The acrylic "button" as described by Moore[10] is a useful

device for preventing contracture of the tracheal stoma following laryngectomy. As shown in Fig. 4-d, the button has an inner flange small enough to allow removal, cleaning and reinsertion by the patient, but large enough to prevent coughing out. The outer flange lies closely against the skin adjacent to the stoma, and is large enough to prevent inadvertent pushing into the trachea with reinsertion.

The "button" may be shaped from heated acrylic tubing by lathe, or by a simpler method in which the flanges are formed by using a length of wooden doweling slightly smaller than the internal diameter (usually three-eighths inch) of the tubing. The dowel is inserted through the tubing as the end is heated to the softening point. Then, with tubing and dowel both held perpendicular to and in contact with a smooth metal surface, the tubing is pushed down firmly to expand the end into a circular flange of the desired size. For the patient who has been wearing a No. 7 tracheal cannula, the diameter of the inner flange should be about 15 mm., and that of the outer flange at least 18 mm. Alterations in diameter of the inner flange may be required for proper fitting in the individual case.

Plastic material (acrylic ?) has been used by Alonso[1] in the treatment of anterior web of the glottis to prevent adherence and recurrence of synechia after endoscopic division. A thin semicircular plate is held in position by sutures brought out above and below the thyroid cartilage until epithelialization is complete.

In the operation recently described by Conley et al,[2] a specially shaped prosthesis attached to a plastic tracheal cannula serves to divert air from the trachea through a mucosa-lined channel into the pharynx for voice production.

Another special variety of acrylic mold used by Erich[3] encircles the outer portion of the tracheal cannula, which is provided with fenestrae. A hollow portion of the mold, projecting upward toward the glottis and communicating with the fenestra below, allowed breathing and speaking in a case in which decannulation could not be accomplished.

SUMMARY.

The properties of "acrylic" (methyl methacrylate) which account for its usefulness in laryngology may be summarized as follows: *1.* High biologic tolerance; absence of tissue reaction is such that molds may be worn for weeks or months with comfort, and without odor or necessity for removal for cleaning. The tendency for contracture following removal is minimal; *2.* Adaptability; by casting of solid molds or heat-shaping of acrylic tubing, apparatus best suited to the given case may be prepared without great effort or inconvenience. The ease with which self-retaining molds may be prepared allows daily changing of the tracheal cannula which, in itself, contributes much to cleanliness of the tracheal fistula and adjacent structures. *3.* Physical characteristics; the absence of softening or change in shape is a desirable feature, and the hard, highly polished surface favors disappearance of granulation tissue and healthy epithelialization.

Flexible molds (rubber or polyethylene) are more easily introduced, but in many instances must be kept in place by retaining sutures or strings. Polyethylene is undobtedly equal to acrylic in tissue tolerance, but it is not easily shaped and, being of softer consistency, lacks optimum smoothness of surface.

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY HOME STUDY COURSES.

The 1959-1960 Home Study Courses in the Basic sciences related to ophthalmology and otolaryngology, which are offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Detailed information and application forms can be secured from Dr. William L. Benedict, executive secretary-treasurer of the Academy, 15 Second Street S. W., Rochester, Minn. Registration should be completed before August 15.

GIANT-CELL TUMOR OF THE SINUSES.*†

LUDWIG A. MICHAEL, M.D.,

Dallas, Tex.

Giant-cell tumors of bone present several controversial features: their origin is not clear, their benign character is subject to increasing doubt, and their response to treatment uncertain.

PATHOLOGY.

Most authors agree in classifying them as true neoplasms, though at times they have been thought to be chronic inflammatory lesions or results of trauma. Jaffe, *et al.*,[7] regard giant-cell tumor as "a neoplasm of a definite kind, arising apparently from the undifferentiated supporting connective tissue of the marrow." Its cytology is typical. The giant cell is the obvious feature from which its name is derived, but the above authors point out that the stromal cells should not be forgotten, as they are really the more significant; indeed, the giant cells are believed to form from fusion of the stromal cells.

Grossly, the lesion involves the epiphysis and adjacent metaphysis of long bones. Further growth and extension are influenced by factors such as trauma by pathological fracture with breakthrough of the periosteum, or the trauma of therapy. Most of the cases fall in the 20 to 40 age group; however, Geschickter and Copeland[5] note that the age incidence of giant-cell tumors of the skull is one decade younger than in the long bones. The majority of the tumors are found about the knee; other sites are the other long bones, vertebrae, anterior portions of the mandible, or maxilla.

Recent literature has shown increasing skepticism of the

*Read at the meeting of the Texas Ophthalmological and Otolaryngological Society, Houston, Tex., Dec. 5, 1958.

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use of the term "benign" as it is applied to this lesion. Much evidence has accumulated which points to a course other than benign, not only after therapy which is presumably incomplete in eradication of the growth, but perhaps *de novo*. Jaffe, *et al.*,[7] introduced grading of this tumor according to cytologic features. Grade I tumors show typical stromal cells, not densely compacted, uniform in size, without abnormal mitoses. Grade II tumors show more atypical stromal cells, but they are not yet quite frankly malignant. Nuclei vary in size and shape, some are multiple, and mitotic figures are frequent and even abnormal. Grade III tumors, though a small group, are frankly malignant and show sarcomatous features. This group more frequently contains tumors which were originally Grade I or II tumors which have become more aggressive over the years. Such a change may either follow irradiation or curettage, or be inherent in the tumor from the beginning. A large group of tumors, formerly called giant-cell tumors, are removed from this category and called giant-cell variants. These include osteofibromas, ossifying enchondromas, or xanthofibromas. Dahlin, *et al.*,[2] called attention to the confusion of giant-cell tumor with nonosteogenic fibroma where the essential difference lies in the fibroblastic activity of the latter. This classification results in a strict delimitation of giant-cell tumors, a designation which, according to some authors, is too sharply circumscribed.[8] At present it seems best to call the lesion giant-cell tumor, benign or malignant, as the circumstances warrant.

DIAGNOSIS AND TREATMENT.

The X-ray appearance and course of these lesions are so characteristic to Brailsford[1] that he feels biopsy is unnecessary and may be disturbing to the lesion. At the other extreme, some feel that a punch biopsy is inadequate to study the lesion and advise removal of enough material for a conclusive pathological diagnosis.

The different forms of treatment used have not been evaluated carefully enough to point to any as definitive. The British, on the whole, recommend X-ray therapy as the treatment of choice, and many American authors subscribe to this

view; on the other hand, in view of recent reports of the effects of radiation, caution must be exercised. There is much evidence that a malignant transformation may follow the use of Roentgen-ray therapy. Sabanas, *et al.*,[11] collected a series of seventeen cases of sarcoma following radiation, of which nine occurred at the site of giant-cell tumors of bone. The average latent period between radiation and onset of sarcoma was over ten years.

Fig. 1. X-ray showing clouding of sphenoid.

Surgical treatment includes radical surgery such as amputation or wide excision, and more conservative methods such as curettage and cauterization. Excision in weight-bearing areas might lead to loss of function, while in other areas it may not be feasible because of inaccessibility. For such tumors, curettage, with or without cautery, and packing with autogenous bone chips can be used. Another method is the combination of radiation and surgery, where a course of radiation is given to reduce the vascularity of the tumor and if necessary curettage is then applicable.

Fig. 2. X-ray showing mass on right with obliteration of landmarks of base of skull.

PROGNOSIS.

Prognosis should be guarded because of the tendency to recurrence and possible malignant transformation. Although it was once believed that these tumors were always benign and remained so, that point of view seems no longer tenable

in the face of reports of malignant transformation and of instances of aggressiveness of the tumor following radiotherapy and repeated curetting.[8,11,12] In a relatively large series of close to 100 cases it was found by Dahlin, *et al.*,[2] that one-half of giant-cell tumors recur after conventional curettage, and ten per cent undergo sarcomatous transformation, some perhaps triggered by radiation therapy.

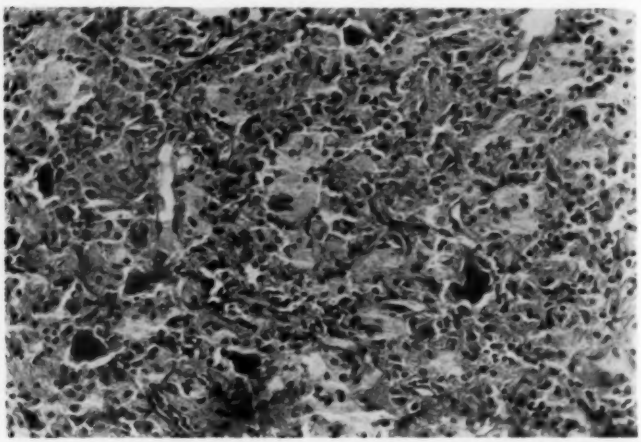

Fig. 3. Giant-cell tumor of bone (X275).

INVOLVEMENT OF THE SINUSES.

Giant-cell tumors of the sinuses are relatively rare and not many have been reported. The diagnosis does not readily suggest itself when found in this location. Geschickter,[4] in a series of 39 benign primary cranial bone tumors found only four giant-cell tumors. Since they are so rare in the sinuses the diagnosis should be made by the removal of tissue for study. Presenting complaints are not characteristic and depend on the site. Proptosis has been common with ethmoid and sphenoid involvement, and pain from nerve involvement has been reported.

Wattles,[13] reviewing the literature in 1937, reported seven

Figs. 4, 5. Lateral X-ray and 8 cm. stratagraph showing calcification in the sphenoid sinus.

cases in the ethmoid and added one. Handousa[6] reported twelve cases, eight of which were in the superior maxilla, three in the ethmoid, and one in the sphenoid. In his series, most patients were females and in the second and third decades of life. His cases did not do well on radiation, and a preference for excision was expressed.

Echols[3] reported a case in the sphenoid with intracranial extension and involvement of the optic chiasma, petrous ridges, middle fossae, and extending to the palate anteriorly and down to the retropharyngeal area. Both surgery and irradiation were used, with a fatal termination.

Peimer,[9] in reviewing the literature in 1954, found reports of thirteen cases in the ethmoids and four in the sphenoids. To these he added three cases, one each in the maxillary antrum and sphenoethmoid and another in the orbital roof. One of these, in which the tumor was excised, did best. Ramamurthi, *et al.*,[10] reported a case of giant-cell tumor of the sphenoid in which extension produced intracranial symptoms. After attempted removal, progression of symptoms was noted, but deep X-ray therapy was refused by the patient. Geschickter and Copeland[5] express a preference for the use of radiation in lesions of the skull, when recurrence following surgery may prove fatal.

To these reports the present author adds one case of giant-cell tumor originating in the sphenoid, treated by X-ray therapy, with remission of symptoms and evidence of healing.

CASE REPORT.

Mrs. C. B., a 16-year-old white married female, was first seen for ear, nose and throat consultation on September 6, 1951. An ophthalmologist referred her for investigation of the presenting complaint of proptosis of the right eye. Her first symptom referable to the present illness was brief periods of numbness over the right infra-orbital area two years previously, accompanied by slight headaches. In August, 1951, she noticed slight proptosis of the right eye accompanied by more severe headaches. These symptoms gradually increased. There was no complaint of nasal discharge, obstruction or bleeding; a slight hearing loss had been noted in the right ear for many years, and there was no change in this symptom.

Physical examination revealed a marked proptosis of the right eye. Nasopharyngoscopy revealed slight encroachment, or enlargement of the anterior lip of the torus on the right. Direct antroscopy through a trocar revealed a clean but shallow right antrum. X-rays showed "a large area of bone destruction extending into the middle fossa and

obliterating the foramen ovale, foramen spinosum, foramen lacerum and extending back almost to the foramen magnum. The destruction extends into the squamous portion of the temporal bone . . . The margins of the bone are smooth; the edges show no motheaten appearance, but are of a normal architecture completely to the edge of the destroyed portion. The area of destruction involves the posterior portion of the floor of the orbit."

On September 14, 1951, at Baylor University Hospital, Dallas, under general anesthesia, the right antrum was entered through a Caldwell-Luc approach. The antrum was found to be normal except that the posterior wall was bulging markedly forward. The bone in this region was intact but thin. When this bone was removed, a smooth, firm encapsulated mass presented. Any manipulation of this mass resulted in severe bleeding. The capsule was entered and thick, cheesy material exuded. Part of the wall was removed for biopsy, the bleeding controlled by cautery, and the operation terminated, leaving the labial incision open.

The biopsy was reported as: "The tissue represents a tumor consisting of a moderate number of multinucleated giant cells surrounded by a loose stroma containing large numbers of round and spindle cells. The giant cells are ovoid shape, with homogeneous, opaque cytoplasm, and contain from six to fifty small oval nuclei. The relative number of the giant cells varies from one place to another. The cells between the multinucleated cells include a large number of foamy macrophages, lymphocytes, and spindle cells with oval vesicular nuclei. In some focal places, the macrophages contain golden granular particles of hemosiderin. The supporting fibrovascular stroma is overshadowed by the cellular elements, and the blood vessels are small and composed of an internal lining of endothelial cells and a thin layer of collagen. No mitoses are seen, and no osteoclastic activity can be demonstrated. The pathological diagnosis is benign giant-cell tumor of bone."

Because of the extent of the tumor no further surgery was attempted. The patient was given external irradiation through a right PA 6 cm. size port and a right lateral 7 cm. size port at 250 KV/15 Ma/½ mm. Cu/1 mm. Al/50 cm. t.s.d., with minimum tumor dose of approximately 1600 *r*, maximum skin dose 3600 *r* in 12 days. Two months later this was repeated; three months later (February, 1952), the third series of external irradiation treatments was given to the tumor through a single right temporal area 5 cm. in size with six equal daily fractions, which added 600 *r* to the minimum tumor dose. The patient did very well. Her eye returned to normal, all symptoms of the lesion receded, and only an oroantral fistula persisted and required occasional irrigation of the antrum through this fistula. One attempt to close the fistula failed, but a second attempt in April, 1956, succeeded. In May, 1955, X-rays revealed "in the lateral view, a particularly excellent demonstration of calcification within the sphenoid sinus. This would indicate that the previous impression of the giant -cell tumor having involved the sphenoid sinus is true. The 8 cm. cut in the lateral stratagraph shows this calcium within the sphenoid sinus well."

The patient was last seen in May, 1957, and was well.

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UNIVERSITY OF ILLINOIS DEPARTMENT OF SPEECH.

As a result of several years of clinical and experimental research, a multiple-choice word list suitable for testing auditory, visual, and combined auditory-visual intelligibility has been developed at the University of Illinois Hearing Center. The recorded auditory test has been found to give rapid and reliable estimates of listening errors. Commercial disc and tape recordings are being prepared for distribution to interested persons. Personnel involved in the development of the test include Charles Hutton, E. Thayer Curry, and Mary Beth Armstrong.

THE INTERMITTENT EAR OCCLUSION TEST.*

A Method of Differentiating Sensations of Hearing and Feeling.

KURT TSCHIASSNY, M.D.,

Cincinnati, Ohio.

Patients with monaural anacousis (complete hearing loss) are frequently unable to differentiate between the acoustic and tactile character of the sensation produced by a vibrating fork on the skull.

Our three cardinal tuning fork tests are frequently not informative for the following reasons:

In the Rinne test it may be easy to confuse the pseudo-negative with the negative Rinne even with masking of the opposite ear, since this maneuver eliminates the acoustic but not the tactile component of the sensation produced by a vibrating tuning fork.

The Schwabach test is not usefull since it provides information concerning only conditions in the opposite good ear. Sound perception may be lengthened, shortened or indifferent depending upon whether there is a conductive, perceptive or no defect at all present.

The significant lateralization toward the "good" ear in the Weber test is frequently missing in these cases, probably due to some as yet unexplained psychesthetic factors.

The following procedure was found to be helpful in identifying unilateral complete deafness and in clarifying the situation wherein the patient is unable to tell whether he "hears" or "feels" the vibrations.

A vibrating tuning fork, preferably 250 or 500 c.p.s., is placed on the patient's vertex or one of his mastoids. The

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examiner places a finger over one of the patient's external meatuses. Now he alternately occludes and opens the orifice. This maneuver when performed in an ear with hearing by air-conduction is accompanied by an acoustic "off-and-on" sensation, distinctly perceived by the patient. This phenomenon can be clearly explained in terms of the classic Weber experiment. The same maneuver, however, when exercised on an ear without hearing by air conduction does not produce any fluctuation of sound, since this sensation depends upon acoustic (otic) but not on tactile factors; therefore, the sensation is reported as a steadily diminishing but not fluctuating sound, since the perception is provided by the opposite, unmanipulated ear.

The rhythm of the "off-and-on" sensation is synchronous with the movements of the finger. As a rule we noticed inward movement to be associated with the "on" sensation while the opposite feeling appears with the release of pressure; however, the reverse effect was obtained upon extremely strong pressure, probably a manifestation of the Gellé principle. This explanation is based on self-observation confirmed by others.

This directional reversal of rhythm is mentioned in order to avoid confusion when the maneuver is first tried. It is the "off and on" sensation, not the directional character of its rhythm, which indicates the presence of hearing.

This test, repeatedly performed by the author, was found to be helpful in differentiating tactile from acoustic sensations produced by a vibrating fork on the head.

It is readily understood that the "fluctuation phenomenon" requires hearing by air-conduction and the presence of adequate ambient noise; therefore, the phenomenon may be absent when sought for in a sound-conditioned room or in patients with severely impaired air conduction, such as in advanced otosclerosis, adhesive processes, radical mastoidectomy, etc. The procedure was demonstrated at the staff meeting of the Department on October 4, 1958. Further studies on a larger scale in cooperation with other members of the Department are in progress.

101 Louis Avenue.

PROGRAM FOR THE 108TH ANNUAL MEETING AMERICAN MEDICAL ASSOCIATION

Section on Laryngology, Otology and Rhinology.

Atlantic City, June 8-12, 1959.

(Rather than assigned discussers, each paper, except those of the Guest of Honor and the Chairman, will be followed by an open discussion period; questions and comments from the floor are encouraged.)

Tuesday, June 9—2 P.M.

PROF. DR. H. H. NAUMANN, Würzburg, Germany—
Intravital Observations of the Nasal Mucous Memberane (Film).

GEORGE M. COATES, M.D., Philadelphia, Pa.—
Address of Guest of Honor.

GORDON D. HOOPLE, M.D., Syracuse, N. Y.—
Modern Otolaryngology.

F. W. DAVISON, M.D., Danville, Pa.—
Laryngeal Obstruction in Children.

BEN T. WITHERS, M.D., Houston, Tex.—
Facility in T&A Management: Conclusions from 2400 Consecutive Cases.

JOSEPH H. OGURA, M.D., St. Louis, Mo.—
Cancer of the Pharynx, Larynx and Upper Esophagus; Surgical Aspects.

BUSINESS MEETING:

Report of Section Delegate to AMA House of Delegates—
Gordon F. Harkness, M.D., Davenport, Ia.
Dean M. Lierle, M.D., Iowa City, Ia., Alternate.

Report of Section Representatives to Board of Governors of American College of Surgeons—
Roderick Macdonald, M.D., Rock Hill, S. C.
Edley H. Jones, M.D., Vicksburg, Miss.
John J. Conley, M.D., New York, N. Y.

Report of Nominating Committee—
F. W. Davison, M.D., Danville, Pa.
Louis E. Silcox, M.D., Philadelphia, Pa.
John D. Singleton, M.D., Dallas, Tex.

Wednesday, June 10—2 P.M.

J. W. HAMPSEY, M.D., Pittsburgh, Pa.—
Current Concepts of Etiology and Management in Otolaryngic Allergy.

VICTOR R. ALFARO, M.D., Washington, D. C.
Address of Section Chairman.

HENRY S. KAPLAN, M.D., San Francisco, Calif.—
New Horizons in the Radiotherapy of Malignant Disease.

JOHN A. KIRCHNER, New Haven, Conn.—
Facial Bone Injuries.

M. R. HIMALSTEIN, M.D., Bay Pines, Fla.—
Obliterative Frontal Sinus Surgery Using Gelfoam.

J. M. RAVID, M.D., New York, N. Y.—
Malignant Melanoma of the Nose and Paranasal Sinuses, and Juvenile Melanoma of the Nose.

Wednesday Evening, June 10—6:30 P.M.

Social Hour and Dinner Honoring the Guest of Honor of the Section, Dr. George M. Coates.

Thursday, June 11—2 P.M.

JOSEPH L. GOLDMAN, M.D., and HARRY ROSENWASSER, M.D., New York, N. Y.—
Current Concepts of the Management of Otitic Infections.

MERLE LAWRENCE, Ph.D., Ann Arbor, Mich.—
Ear Physiology.

EDWARD W. HARRIS, M.D., Columbus, O.—
Symptoms Referable to the Eustachian Tube.

GEORGE KELEMEN, M.D., Boston, Mass.—
Maternal Diabetes and Congenital Deafness.

JOSEPH SATALOFF, M.D., Philadelphia, Pa.—
Stapes Mobilization with Long-Standing Otosclerosis.

HOWARD P. HOUSE, M.D., Los Angeles, Calif.—
Polyethylene in Middle Ear Surgery.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

The Annual Meeting of the Pacific Coast Oto-Ophthalmological Society will be held May 3-7, 1959, at Hotel Riveria, Las Vegas, Nevada.

A very superior Scientific Program has been arranged for the Las Vegas meeting. We have an outstanding group of guest speakers in addition to our participating members. A complete day of scientific programs has been arranged for Monday with the guest speakers participating in the General Session in the morning in the Clover Room. The first half of the Instruction Courses will be given Monday afternoon, and the second half of the Instruction Courses will be given Tuesday morning. The Instruction Courses are being given in many instances by the invited guest speakers as well as our own members. Many of the courses will have an interest for both eye and ear, nose and throat men this year.

The Men's Luncheon will be held Monday noon in the Convention Hall of the Hotel Riveria.

The Social Events Committee has planned an interesting social program for the members and their wives during the time of the meeting. Two dinner show activities are planned: Monday night at Hotel Sahara; the second dinner show will be the Annual Banquet held at the Hotel Riveria Wednesday night. Tuesday evening is to be free and a reservation desk to help members obtain other show reservations will be maintained at the Registration Desk. The golfers will use the Las Vegas Municipal Golf Course for their tournament. There is to be no fishing derby this year. An interesting sightseeing tour of Boulder Dam has been planned.

The President's Reception and Buffet Supper will be held Sunday evening, May 3, on the patio of the Hotel Riveria.

The Ladies Luncheon will be held on the patio of the Hotel Riveria, and will include an interesting fashion show.

It may not be possible to accommodate everyone in the Hotel Riveria due to the room limitation. Space in the Hotel Riveria will be assigned in order of the reservations received by the Hotel. When the space has been filled, reservations will be referred to the Hotel Sahara.

The Hotel Riveria is to be the headquarters of the meeting. Write directly to the Hotel Riveria for all room reservations.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Erling W. Hansen, 90 So. Ninth St., Minneapolis, Minn.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 10-15, 1959.

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Council: Savoy Plaza, New York, N. Y., January 10, 1959.
Middle: Park Plaza Hotel, St. Louis, Mo., January 12-13, 1959.
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Annual Meeting: Chicago, Ill., October, 1959 (definite time and place to be announced later).

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Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Eighth International Congress of Bronchoesophagology, Mexico City, March 14-18, 1959.

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Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

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Meeting: Seventh Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Miami, Fla., March, 1960.

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